













# SURGERY FOR DENTAL STUDENTS

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SCIENTIFIC PUBLICATIONS

BLACKWELL

FIRST PUBLISHED 1921

*Printed in Great Britain in the City of Oxford by*

A. R. MOWBRAY & CO. LIMITED

*and bound by*

THE KEMP HALL BINDERY

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## FOREWORD TO THE FIRST EDITION

I have read the manuscript of this book with profound interest and admiration. I am convinced that it will prove extremely popular because it fulfils the two great needs of the dental student: it presents the principles of general surgery in an attractive and easily assimilated manner, and provides all the information required for the qualifying examination.

General surgery is often regarded by the dental student as an added burden to a heavily-loaded curriculum. It is nothing of the sort. As Professor Woodruff says in his Introduction, dental surgery is but a specialized branch of general surgery, and no specialist can practise his art unless he has had a thorough grounding in general surgical principles. The cardiac surgeon and the neuro-surgeon, who often steal the headlines, could not have achieved their eminence without a training in general surgery. The dental surgeon cannot assume responsibility for his patients unless he is familiar with the tissue changes which accompany inflammation, injury, repair and tumour formation; he is a general surgeon practising dentistry.

The examination of dental students in general surgery is designed to test their knowledge of basic principles and their ability to apply these principles to the diagnosis and treatment of the common diseases of the mouth and jaws. To satisfy the examiners the student must show evidence of a proper training in clinical surgery, and to supplement his practical experience he requires a suitable textbook. Professor Woodruff has written his book to meet this need and has, in my opinion, succeeded.

This is not a textbook of Dental Surgery. It is an account of the principles of general surgery illustrated wherever possible by applications of particular interest to the dental student. It covers the whole field of regional surgery normally specified in an examination syllabus. The wisdom and knowledge of an experienced teacher have been put into the pages which follow; they will serve to arm the dental student both for his present needs and the subsequent practice of his art.

S. H. WASS



## PREFACE

The second edition follows the same general plan as the first, but the book has been brought up to date and many sections have been completely rewritten.

I am grateful to Professor A. Amies and Professor F.A. Craddock for many helpful suggestions, and to Professor J.I.P. James, Mr A.B. Wallace, Mr A. Brownlie-Smith and Dr Howard Davies for reading the sections relating to orthopaedics, plastic surgery, oto-rhino-laryngology and blood clotting respectively. In addition Professor James has provided me with two new illustrations (Figures 20 and 21).

Figure 27 is reproduced from an article in the *British Journal of Radiology* by Dr William Campbell, and for this I am most grateful both to the author and to the editor of the journal. Figures 34 and 35 have been redrawn by Mr C. Shepley.

I am especially indebted to Dr W. Donald MacLennan, who has devoted much time to helping me to redraft the more specifically dental sections of the book and has kindly provided figures 22, 23, 24, 29 and 30.

As in the preface to the first edition, in expressing my thanks to these friends for their candid and helpful criticism I hasten to add that I have not always taken their advice and they cannot be held responsible for any of the opinions expressed.

Finally, my thanks are due to Mrs E. Robertson and her colleagues for typing revised sections of the book, to Mr Alan Dean who has kindly read the proofs, and to Mr Per Saugman of Blackwells for his encouragement and co-operation.

### **From the Preface to the First Edition**

This book is based on a course of lectures to dental students delivered in the University of Sheffield. In writing it I have been very conscious of the debt I owe to my own teachers and senior colleagues, especially Mr A.E. Coates, Professor R. St.Leger Brockman and Professor W.C. Wilson.

I am deeply grateful to Mr S.H. Wass for the interest he has shown in the book and for his wise guidance at all stages of its preparation. He has



read the entire manuscript with great care and has suggested many important alterations; in addition he has very kindly contributed a Foreword. Dr Hugh Crombie has read the more specifically dental sections of the book, Mr C.D. Weir the section on inflammation in the nose and accessory nasal sinuses and Dr Ernest Ridley the section on radiotherapy.

I am much indebted to Dr Warren Cole, Chairman of the Department of Surgery, University of Illinois, for Figures 78 and 88; to Professor C.G. Rob and the photographic department, St Mary's Hospital, for Figures 38 and 47; to Mr G.W. Blomfield and the staff of The Graves Institute of Radiotherapy, Sheffield, for Figures 61 and 62; to Mr Wilfrid Hynes for Figures 10, 11 and 35; to Mr Charles Donald and the photographic department of the Hospital for Sick Children, Great Ormond Street, for Figures 14 and 81; to Dr Ambrose King and the Whitechapel Clinic for Figure 49; and to Mr S.H. Wass and the photographic department, Guy's Hospital, for Figures 13, 46, 50, 59, 68, 69, 70 and 100. The copyright of all these illustrations remains the property of the individuals or institutions mentioned.

Figure 90 is a photograph by Mr Cain of two specimens belonging to the Department of Anatomy, University of Aberdeen, and is reproduced by permission of Professor R.D. Lockhart; Figure 91 has been kindly provided by Dr T.N. Morgan; Figures 53, 55, 56, 60 by Mr J.F. Philip; Figure 43 by Dr A. Ross; Figure 77 by Mr J.J. Brownlee; and Figures 83 and 84 by Professor E.S.J. King and Mr R. Inglis.

All the other photographs have been taken in the photographic department of the Medical School, University of Aberdeen, by Mr R.G.M. Drummond. In thanking him for his skill and care I should like also to express my thanks to Dr D.P. Levack for the loan of radiographs and to various colleagues at the Aberdeen Royal Infirmary who have allowed me to have photographs taken of their patients. For the drawings I am indebted to Mr D. Emslie, who has prepared them from rough sketches of my own.

Finally, my thanks are due to Miss G.M. McCallum, who has typed successive drafts of the manuscript with unfailing patience and accuracy, to Professor J.P. Walsh who has kindly read the proofs and to Mr John Grant for his never-failing help and co-operation.

## INTRODUCTION

There are four main reasons why a course of training in surgery is of value to dental students.

In the first place, dental surgery, as its name implies, is a branch of surgery in the general sense of the word. Like other specialized branches, dental surgery necessitates a highly specialized training, but this training must be based on a sound knowledge of fundamental surgical principles. These principles must first be considered in relation to the wider realm of general surgery; only then can their application to some specialized branch such as dental surgery or oto-rhino-laryngology be properly understood.

Secondly, cases occur in dental practice in which the differential diagnosis between a dental condition and a surgical condition (in the narrow sense of the word) has to be made. For example, the pain of trigeminal neuralgia may have to be distinguished from pain due to dental sepsis; and the trismus of tetanus from that associated with an impacted molar tooth. When doubt exists a dental surgeon should consult a surgical colleague, but it is obvious that he can do this only if he is aware of the difficulty.

Thirdly, a dental surgeon who has some knowledge of the regional surgery of the head and neck may, in the course of his examination of a patient, observe some purely surgical lesion, such as an early epithelioma of the lip or septic tonsils, and be able to refer the patient to a surgeon for treatment without delay.

Finally, there are some patients—for example, those with fractures of the jaw—who may require the combined services of a surgeon and a dentist. In such cases it is obviously a great advantage if those responsible for the treatment have an intelligent appreciation of each other's work.

It is the opinion of the writer that the first of these reasons is by far the most important. If this is correct the primary object of a course in surgery for dental students should be to teach surgical principles. It follows that the usual plan of presenting the dental student with an elementary treatise on the regional surgery of the head and neck, hoping that he will perceive the underlying general principles for himself, is mistaken. What is needed is an account of the basic principles of surgical diagnosis and treatment, and the clinical and pathological features of surgical conditions which are

not limited to one particular region of the body, illustrated wherever possible by reference to the common surgical disorders of the head and neck.

The present book is intended to meet this need. At the same time attention has been paid to examination requirements and it has been found possible to include in the examples virtually all the regional surgery specified in the syllabus for the Licentiate in Dental Surgery of the Royal College of Surgeons of England. The student who has read the book in conjunction with a proper course of clinical instruction should therefore find no difficulty with the surgical part of this examination or of the qualifying examination for a university degree in dental surgery.

## *Chapter I*

# SURGICAL DIAGNOSIS

Almost every patient who consults a surgeon has some definite complaint; for example, pain, bleeding, a localized swelling, an ulcer, retention of urine, limitation of movement of a joint and so on. In seeking professional advice he has two main objects: first, and most important, he wants *treatment*; secondly, he wants a *prognosis*—that is, advice as to the probable consequences of his disorder.

It is the task of the surgeon to satisfy, as far as he can, both these requirements, and to achieve this he must first of all make an accurate *diagnosis*.

In making a diagnosis reliance is placed mainly on the patient's history and the results of clinical examination, but when these are insufficient various special investigations must be undertaken. The art of taking a reliable history and making an accurate physical examination can be acquired only by practice, but attention may usefully be drawn to certain points which are of special importance.

### **The History**

Note first the name, age, sex, marital state and occupation of the patient. Find out the nature of his complaint and its duration, recording the patient's story as far as possible in his own words. Finally, make a systematic inquiry, asking relevant questions suggested by the patient's account of his symptoms and, when necessary, including questions about the family history.

Be tactful but persistent, and try to avoid asking leading questions.

### **The Clinical Examination**

The clinical examination should proceed according to a definite plan. In surgical practice it is often best to begin with a preliminary general examination of the patient and then examine the local lesion thoroughly. Finally complete the general examination in as much detail as the circumstances require.

### Preliminary General Examination

Inspect the patient, noting his general state of health, his posture, his colour, the rate and rhythm of his respiration, and the appearance of his eyes, face and hands. Feel his pulse and count the pulse rate. If he is in a hospital ward examine the temperature chart hanging above his bed.

### Examination of the Local Lesion

Detailed accounts of local lesions—of the skin, bones, joints, lymph nodes and so on—will be given in succeeding chapters. Here we are concerned only with methods of examination, and to illustrate these we shall describe the general procedure for the examination of a localized swelling, and the systematic examination of the mouth and throat.

#### Examination of a localized swelling

In every case a twofold diagnosis—anatomical and pathological—has to be made. There are thus two questions to be answered:

First, *where* is the lesion, or more precisely, in what anatomical structure or tissue does it arise?

Secondly, *what* is the nature of the underlying pathological change?

Students are apt to neglect the anatomical diagnosis but it is of great importance and, as we shall see presently, often provides a valuable clue to the pathological diagnosis.

The methods used are primarily *inspection* and *palpation*, assisted when necessary by *percussion*, *auscultation*, *transillumination* and *measurement*.

With all these methods the *principle of comparison* is used whenever possible; that is to say, a diseased part, such as a limb, eye or breast, is compared with the corresponding healthy member on the opposite side.

#### *Inspection*

Inspection is of great importance. It causes the least possible discomfort to the patient, and often yields information which cannot be obtained in any other way. Signs such as pigmentation, dilatation of superficial veins and dimpling of the skin, for example, may be of great diagnostic significance and can be recognized only by inspection. Moreover, some swellings are more easily seen than felt and may be missed completely if inspection is omitted.

### Palpation

Palpation is defined as examination with the hands. The term means much more than simply passing one's hands over the swelling; it implies a deliberate attempt to elicit physical signs, and includes the performance of certain special tests. Palpation helps to confirm the results of inspection and provides additional information which cannot be obtained by the eye alone.

In palpating a swelling proceed as follows:

Determine first the size, shape and consistence of the swelling. Note if it is tender, if it pulsates, if there is any local change in temperature and if crepitus is present. Finally, test for fluctuation, pitting on pressure, and fixity of the swelling to the skin and to deeper structures.

It is necessary to explain some of these terms and describe how the various tests are performed.

*Pulsation.* Pulsation is of two kinds, *transmitted* and *expansile*. In the former case the whole swelling moves *en masse* as a result of pulsation in some nearby structure, usually a large artery. In the latter case the swelling itself expands and contracts synchronously with the heart beat. This implies either that the swelling is an aneurysm or that it is extremely vascular, as, for example, some thyroid swellings.

The distinction between the two types of pulsation can usually be made by careful palpation.

*Local changes in temperature.* A local increase in temperature is characteristic of acute inflammation (Chapter X).

Local coldness is unlikely to be confined to a swelling but may affect some part of the body such as a finger, hand, foot or limb. The significance of this sign is discussed in Chapter XVII.

Local changes in temperature may be estimated by palpation, preferably with the back of the hand. If an accurate determination is required the skin temperature must be measured with a thermo-couple.

*Crepitus.* Crepitus is a peculiar grating or purring sensation which may be felt on simple palpation or when a nearby joint is moved. Crepitus may signify:

1. The presence of gas in the tissues. This condition is termed *surgical emphysema* and may be due to gas gangrene (Chapter XII) or to injury, especially of the lung or larynx.
2. Arthritis (Chapter XX).
3. Inflammation in a tendon sheath.
4. A fracture (Chapter VI).

**Fluctuation.** Fluctuation is characteristic of cystic swellings; that is, swellings which contain fluid (Chapter XV). The test for fluctuation may be carried out with the two index fingers, or with the index and middle fingers of one hand and the index of the other. In the latter case the procedure is as follows:

Place the index and middle fingers of one hand (the 'watching fingers') midway between the centre of the swelling and the periphery, and apply pressure to the centre of the swelling with the opposite index finger (Fig. 1).



FIG. 1. Testing for fluctuation. The lesion is an abscess (Chapter X) overlying the manubrium sterni.

If the swelling is mobile it must be fixed; this can usually be done with the watching fingers, but occasionally an assistant is needed. Make the test in two directions at right angles. Fluctuation is present if pressure is transmitted to the watching fingers *in both instances*.

Fluctuation is usually a reliable sign though it is occasionally absent in tense cysts. A soft solid swelling such as a lipoma sometimes appears to fluctuate but can usually be distinguished from a truly cystic swelling.

**Pitting on pressure.** Pitting on pressure indicates the presence of *oedema*; that is, accumulation of fluid in the subcutaneous tissue. Widespread oedema may occur in cardiac failure or chronic renal disease, but is of interest mainly to the physician. More localized oedema may result from venous or lymphatic obstruction (Chapters XVII and XVIII). It may also be seen over an area of deep-seated suppuration (Chapter X).

To test for pitting make firm pressure with a finger at the suspected site. The test is positive if a pit or depression is formed and remains for some time—usually a few minutes—after the pressure is released (Fig. 2).



FIG. 2. Testing for pitting oedema. The patient is suffering from congestive cardiac failure.

*Fixity to skin.* If a swelling is fixed to the skin it must either arise in the skin or have become attached owing to extension of the pathological process. A common example of a swelling arising in the skin is a sebaceous cyst (Chapter XV). Secondary attachment to the skin often occurs with malignant tumours (Chapter XIV) and in various inflammatory conditions (Chapters X and XIII).



To test for fixity to the skin fix the swelling with one hand and gently move the skin over it with the other. Even slight degrees of fixity are detected because the skin dimples at the point of fixation (Fig. 3). This is a much more sensitive test than the method of picking up the skin over the swelling with the finger and thumb.



FIG. 3. Testing for fixity of a swelling to the skin. The patient had noticed a lump in the breast and some retraction of the nipple. On gently moving the tumour the skin dimpled at a point about an inch above the nipple. The lesion is a carcinoma (Chapter XIV).

*Fixity to deeper structures.* To test for fixity to a muscle see whether the mobility of the swelling is reduced when the muscle is contracted (Fig. 4). To test for fixity to a structure which is itself mobile inspect and palpate the swelling while the patient makes appropriate movements. For example, if a swelling is thought to arise in the thyroid gland ask the patient to drink a mouthful of water. The thyroid is attached to the larynx and therefore moves upward with this structure during swallowing.



FIG. 4. Testing for fixity of a swelling to an underlying muscle. The patient has an advanced carcinoma of the breast. When the pectoralis major muscle was made to contract by the manoeuvre shown the mobility of the swelling was greatly reduced.

### Other methods of examination

*Percussion* is used in the examination of abdominal swellings and *auscultation* may help to confirm the diagnosis of aneurysm.

*Transillumination* is often useful in the case of swellings in the neck (*vide infra*) and scrotum. Use a pocket torch and determine whether or not light is transmitted through the swelling. In doubtful cases repeat the test in a darkened room or observe the swelling through a tube made by rolling up a sheet of paper.

Measurement, repeated at intervals, is important when one wishes to determine whether a swelling is increasing or decreasing in size.

By the methods enumerated the physical characteristics of the swelling—its size, shape, colour, consistence, position, mobility, sensitivity, vascularity and other special features—are determined, and its anatomical relationships are ascertained. Then and only then should the pathological diagnosis be considered.

There are many lesions—for example, a hare lip, a black eye, Hutchinson's teeth or an extensive carcinoma of the tongue—which one learns to diagnose instantly; in Sir Thomas Lewis's phrase, one recognizes them 'as one recognizes a dog's bark'.

Where the pathological diagnosis is not obvious the first step is to attempt to classify the lesion under one of the following etiological headings:

1. Congenital.
2. Traumatic.
3. Inflammatory (acute or chronic).
4. Neoplastic (benign or malignant).

This scheme is a useful one, but it must be remembered that it is not always applicable. It omits, for example, lesions due to dietetic deficiency and various degenerative conditions, and there are a number of conditions whose correct classification is still in doubt.

### *Examination of a swelling in the neck*

Much can be learned from inspection. The diagnosis of a superficial swelling such as a sebaceous cyst (Chapter XV) or a pigmented mole (Chapter XIV) is usually obvious. If the swelling is more deeply situated observe first whether or not it is in the mid-line. If mid-line it is probably situated in the thyroid gland (Chapter XXII) or arises from remnants of the thyroglossal duct (Chapter III), though it may be a dermoid cyst (Chapter XV). Ask the patient to swallow; if the swelling moves upward the diagnosis of a thyroid or thyroglossal swelling is confirmed. If the

swelling is situated laterally it is probably arising from lymph nodes (Chapter XVIII), the submaxillary salivary gland or the lateral lobe of thyroid; alternatively it is a branchial cyst (Chapter III) or a cystic hygroma (Chapter XVIII); or less commonly an aneurysm (Chapter XVII) or a tumour of the carotid body.

To palpate the neck begin by standing behind the patient, placing the hands as shown in Figure 5. If a swelling can be felt proceed to examine it in detail as already described; note especially whether it is fixed to skin



FIG. 5



FIG. 6

FIG. 5. Palpation of the neck.

FIG. 6. Determining the relation of a swelling to the sterno-mastoid. The muscle has been made to contract by the manoeuvre shown. The swelling is a mass of tuberculous lymph nodes (Chapter XIII) lying partly beneath and partly anterior to the muscle.

or deeper structures, whether it moves when the patient swallows and whether it fluctuates or pulsates. Determine whether or not the swelling is wholly or partly deep to the sternomastoid by palpating when this muscle is strongly contracted (Fig. 6). Finally, palpate bimanually with one finger in the mouth (Fig. 7). Swellings of the submaxillary salivary

gland or of the lymph nodes closely related to it can be felt easily in this way.

If the swelling is cystic try transillumination; a cystic hygroma is usually brilliantly translucent.

If it seems possible that the swelling arises from lymph nodes, examine carefully the territory drained by the nodes (Chapter XVIII), not forgetting the lips, mouth and tongue in appropriate cases.



FIG. 7. Bimanual palpation of the floor of the mouth.

### **Examination of the mouth and throat**

Begin with the lips. Inspect them, looking particularly for cracks and ulcers, and if any lesion is seen palpate the lips, using a glove if there is reason to suspect a chancre.

Examine next the cavity of the mouth. If the patient wears dentures ask him to remove them. With the help of a pocket torch and a spatula, inspect systematically the inner aspect of the cheeks, the gums and teeth, the tongue, the palate and the floor of the mouth. Look particularly at the orifices of the parotid (Stenson's) ducts on the inner aspect of the cheeks opposite the second upper molar teeth and the orifices of the submaxillary (Wharton's) ducts in the floor of the mouth on each side of the frenum.

Ask the patient to put out his tongue and note whether there is any asymmetry of this organ and whether it deviates from the mid-line when protruded.

Depress the tongue with a spatula and inspect the back of the throat and the tonsils (Fig. 8).

Then palpate any obvious lesion, using a glove when necessary, and feel bimanually the floor of the mouth. Test for fluctuation and transillumination in appropriate cases; a swelling in the floor of the mouth

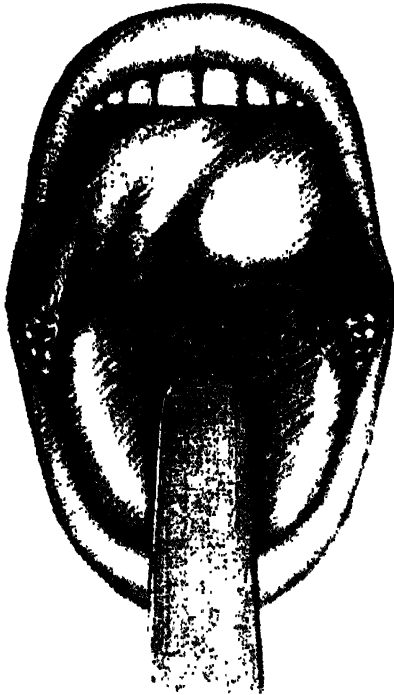


FIG. 8. Examination of the throat. The patient is suffering from quinsy (Chapter X).

giving a positive result to both tests would almost certainly be a ranula (Chapter XV).

Finally, examine the submental, submaxillary and jugular lymph nodes.

### **Detailed General Examination**

In trivial cases this is sometimes omitted. Before administering a general anaesthetic, however, always examine the patient's heart and chest, and test his urine for sugar and albumen.

In serious cases a thorough examination must be made of the cardiovascular and respiratory systems, the abdomen and the nervous system by the anaesthetist or a consultant physician.

### **Special Investigations**

Some special investigations may be undertaken single-handed, others require the help of a colleague. Examples of both kinds are given below.

#### **Examination of interior parts of the body with specially constructed illuminated tubes**

The instruments used include the laryngoscope, bronchoscope, oesophagoscope, gastroscope, sigmoidoscope and cystoscope. All save the gastroscope are completely rigid. Usually either local or general anaesthesia is required for the examination.

Examination of the larynx with the laryngoscope is called direct laryngoscopy. An alternative way of examining the larynx is to use a mirror held against the patient's soft palate and a headlamp. This is called indirect laryngoscopy.

#### **Microscopic examination of body fluids and excreta**

Microscopic examination of blood, urine, faeces, cerebro-spinal fluid and material aspirated from swellings is often of diagnostic value. It may, for instance, be difficult to distinguish on clinical grounds alone between breaking down tuberculous cervical glands and a branchial cyst, but aspiration of some fluid from the swelling quickly settles the matter. In the former case typical tuberculous pus (Chapter XIII) is obtained; in the latter the fluid has a shimmering appearance to the naked eye, and under the microscope is seen to contain epithelial cells and numerous cholesterol crystals.

#### **Bacteriological investigation**

The materials mentioned in the last section, and also discharges from wounds and sinuses, are commonly sent for bacteriological examination. Three types of investigation may be undertaken: (a) microscopic examination of direct smears, (b) culture and (c) animal inoculation. Great care must be taken to avoid contamination of the specimen when culture or animal inoculation is contemplated.

#### **Biochemical investigations**

Biochemical investigations are of many kinds. Common examples are examination of urine for sugar and albumen, and quantitative estimation of blood sugar and blood urea.

**Radiological examination**

Radiology was originally restricted to examination of bones and joints, but is now applied to many other structures.

*Bones and joints*

In every case of suspected injury or disease involving a bone or joint radiographs should be taken in two planes at right angles (usually antero-posterior and true lateral). In some cases additional oblique views are required and sometimes, especially in the radiology of the skull, stereoscopic films are taken.

*Teeth*

Intra-oral films are widely used to reveal cavities, root abscesses, unerupted teeth, and cysts and tumours of dental origin (Chapter XV). The technique of taking such films is described in textbooks of Dental Surgery.

*Soft tissues*

Ordinary radiographic methods are important in the diagnosis of many soft tissue lesions, especially in the lungs. They also reveal deposits of calcium, some calculi (Chapter XXIII) and many foreign bodies.

Special techniques are used to show up hollow structures. These include (a) direct introduction of a radio-opaque substance; (b) administration of a radio-opaque substance which is subsequently excreted into the cavity; and (c) introduction of air which allows the surrounding soft tissues to be shown up by contrast. Some of these techniques are listed in Table I; *it is intended for reference only*.

**Examination of biopsy specimens**

Biopsy means removal of part or sometimes the whole of a lesion for examination by a pathologist. Naked eye examination of the specimen is occasionally conclusive, but in most cases sections must be cut and examined under the microscope.

The procedure is commonly applied to swellings of lymph nodes and to tumours of various kinds. Ordinarily paraffin sections are prepared and a report is sent about 48 hours after removal of the specimen; in special cases frozen sections are made and a report can be obtained in a few minutes. Paraffin sections give more reliable results and whenever frozen sections are made paraffin sections should be prepared later to check the diagnosis.

TABLE I. Some 'Special' X-ray examinations. For reference only.

Name	Structures shown up by the procedure	Substance used	Method of administration	Usual object of the examination
No special name	Sinuses or fistulae of various kinds	Diodrast or similar substances	By needle or catheter introduced into the sinus or fistula	To demonstrate the extent of a sinus or fistula (see Chapter XVI)
Sialogram	Parotid and sub-maxillary glands and ducts	Diodrast or similar substances	By fine catheter introduced through the duct	The diagnosis of diseases of the salivary glands (see Chapter XXIII)
Bronchogram	Bronchi	Dionysil	By catheter in the trachea or a bronchus	Diagnosis of bronchiectasis and carcinoma of the lung
Myclogram	Spinal canal	Myodil	By lumbar puncture	To demonstrate spinal tumours and other lesions causing compression of the spinal cord and nerves, and to determine the precise level of the lesion
Barium swallow	Oesophagus	Barium suspension	Orally	Used in cases of dysphagia to see if there is any oesophageal obstruction and, if so, to help in determining the site and cause (e.g. carcinoma, fibrous structure, cardio-spasm—see Chapter XXIII)
Barium meal	Stomach, duodenum and sometimes intestine	Barium suspension	Orally	The diagnosis of peptic ulcer of the stomach and duodenum and carcinoma of the stomach



TABLE I—*continued*

Name	Structures shown up by the procedure	Substance used	Method of administration	Usual object of the examination
Barium enema	Colon	Barium suspension	As an enema	The diagnosis of carcinoma and other lesions of the colon
Venogram and arteriogram	Veins and arteries	Diodrast or similar substances	By needle into a vein or artery	To demonstrate obstruction of a vein or artery (see Chapter XVII)
Pyelogram (retrograde)	Kidney (renal pelvis) and ureter	Sodium iodide or Diodrast	By catheter inserted through a cystoscope into the ureter	The diagnosis of many lesions of the urinary tract
Pyelogram (intravenous)	Kidney (renal pelvis), ureter and bladder	Diodrast	Intravenous	The diagnosis of many lesions of the urinary tract
Cholecystogram	Gall bladder	Tetra-iodo phenolphthalein	Usually orally	The diagnosis of chronic cholecystitis and cholelithiasis (gall stones)
Cholangiogram	Bile ducts	Biligratin	Intravenous	To demonstrate lesions of the bile ducts including stones
Encephalogram and ventriculogram	Ventricles of brain	Air	By lumbar or ventricular puncture	To demonstrate and localize tumours and other lesions in the brain
Arthrogram	Joints	Air	By needle introduced into the joint	To demonstrate certain soft tissue injuries of joints (e.g. displacement of a semi-lunar cartilage in the knee). (Chapter VII)

The removal of biopsy specimens is usually a minor procedure and is normally done under local analgesia. Two methods are in common uses (a) removal of a piece of tissue under vision through a small incision; (b) 'blind' removal of tissue through a wide-bore needle. The latter method is especially simple from the patient's point of view, but is only effective with certain types of tissue.

**Exploratory operation**

Exploratory operation is an extension of the method described in the last section. It is often a major procedure and should only be undertaken when absolutely necessary. The diagnosis may be obvious when the lesion is exposed; sometimes, however, it remains in doubt until the whole or part of the lesion has been removed and examined microscopically. Whenever possible a curative operation should be performed at the same time as the exploration.

## *Chapter II*

# THE PRINCIPLES OF SURGICAL TREATMENT

The methods of treatment employed in surgical practice cover a wide range. They may, however, be conveniently classified under six main headings:

1. General measures.
2. Administration of drugs.
3. Physiotherapy.
4. Radiotherapy.
5. Manipulative surgery.
6. Operative surgery.

### General Measures

General measures include rest, keeping the patient warm, regulating his bowels, controlling his smoking and consumption of alcohol, administering fluids and providing a suitable diet. Some of these require further consideration.

#### Rest

This term includes both *general rest* and *local rest*. General rest means that the patient's activities are limited in various ways; he may, for example, be put to bed, or he may simply be kept off work or strenuous exercise. Local rest means that a particular part of the body is rested; a limb may be splinted, an eye bandaged to exclude light or a lung collapsed by inducing a pneumothorax (Chapter XIII).

#### Administration of fluids

Fluid is continually being lost from the circulation, both *externally*, as in sweat and urine, and *internally* to the tissues. In normal people the loss is balanced by fluid ingested or reabsorbed from the tissues, and the blood volume remains fairly constant.

The fluid loss may be increased as a result of injury or disease; the external loss, for example, may be increased by vomiting, exudation from wounds, or haemorrhage, and the internal loss is increased in patients with severe burns (Chapter IX). In such cases a correspondingly large fluid

intake is necessary to avoid a serious fall in blood volume. When possible this extra fluid is given by mouth; but other routes must be used if the patient is unable to swallow or is vomiting, if the blood volume has already fallen appreciably and rapid restoration is required, or if it is necessary to replace plasma or whole blood.

At one time water was often given per rectum, and saline subcutaneously, but nowadays intravenous administration is routine, and is essential when plasma or blood is to be given.

✓ The guiding principle in administering fluid is to make good the patient's deficiency, both *qualitatively* and *quantitatively*. If the patient has had a severe haemorrhage he is given whole blood of appropriate group (Chapter VIII); if, as a result of burns, he has lost water, salt and proteins, he is given plasma. If he has lost only water and salt he is given sufficient normal saline (0.9 per cent NaCl) to make good the salt deficiency and such additional water as is necessary in the form of 5 per cent glucose solution (the glucose being included to increase the osmotic pressure of the fluid and to act as a source of energy). If insufficient fluid is given the desired effect is not achieved; if too much is given cardiac failure, oedema of the lungs and death may result. If he is deficient in potassium this is replaced in the form of potassium chloride. It is difficult to estimate the amount of potassium required because, in the body, potassium, unlike sodium, is mainly intracellular, and if too much is given death may occur from cardiac arrest; as a general rule therefore potassium should be given slowly and cautiously, and only if the patient is passing adequate quantities of urine.

### **Administration of Drugs**

A drug may be given to produce a general effect or a local effect.

To produce a general effect the drug may be given orally (either swallowed or dissolved under the tongue), or by subcutaneous, intramuscular or intravenous injection. The choice of route is determined largely by the nature of the drug and the rapidity of action required. The mode of administration of various antibiotics is discussed in Chapter XI. Morphine may be given by any of the routes mentioned; oral administration produces the slowest effect and intravenous administration the most rapid, but in most cases subcutaneous injection is used.

Drugs used to produce local effects include local anaesthetic agents, sclerosing fluids used in the treatment of varicose veins (Chapter XVII), antibiotics suitable for topical use (Chapter XI).

### **Physiotherapy**

Physiotherapy includes exercises, massage and irradiation with ultra-violet or infra-red rays.

Two types of exercise are used, *active* and *passive*. Active exercises are carried out by the patient himself, and the function of the surgeon or physiotherapist is to provide encouragement and see that the patient does not attempt too much. In most cases active exercises are safe if they do not cause pain.

Passive exercises are those in which a joint is moved passively by the surgeon. They are less useful than active exercises and decidedly less safe.

### **Radiotherapy**

Until recently the term radiotherapy meant treatment by means of radium, radon or X-rays; it now includes in addition treatment by a wide range of radio-active isotopes.

Radiotherapy finds its main application in the treatment of malignant neoplastic disease. Often a choice has to be made between radiotherapy and operative surgery; the surgeon should therefore be aware of the scope and limitations of both forms of treatment and the degree of discomfort each may entail for the patient. This subject will be discussed in Chapter XIV.

### **Manipulative Surgery**

Manipulative surgery is commonly employed by orthopaedic surgeons. It is also used, sometimes usefully, often pointlessly, and sometimes dangerously, by unqualified 'bone-setters'.

The main use of manipulative surgery is in the reduction of fractures (Chapter VI) and dislocations (Chapter VII), in the correction of deformities such as congenital club-foot, and in the treatment of certain types of stiff joint (Chapter XX).

In some cases no anaesthetic is necessary; in others a general or a local anaesthetic is used. The manipulation may be carried out entirely by hand or with the help of some kind of apparatus. Following manipulation a splint or other form of retentive apparatus is often applied.

### **Operative Surgery**

A surgical operation implies the deliberate infliction of a wound for purposes of treatment. For the patient this procedure is always unpleasant

and sometimes hazardous, but both the discomfort and the risk have been greatly reduced as the result of advances along three main lines:

1. The introduction of anaesthetics (Wells and Morton, 1844; Sir James Simpson, 1847) and the subsequent developments in anaesthetic methods.
2. Improvements in surgical technique, especially the introduction of antiseptic methods (Lister, 1865) and the subsequent development of asepsis.
3. Improvements in methods of pre-operative and post-operative treatment.

### **Types of operation**

Hundreds of different operations have been devised but most of them fall under one or more of the following headings:

1. Incision and drainage.
2. Creation of a fistula.
3. Removal of diseased or abnormal structures.
4. Interruption or excision of normal structures.
5. Operations for the arrest of haemorrhage.
6. Operations to provide local rest to a diseased or injured part.
7. Reconstructive operations.

#### *Incision and drainage*

Simple incision is commonly undertaken to evacuate pus from an abscess. In such cases the incision is not sutured, but is left widely open, and sometimes a piece of rubber sheet or tubing is inserted to assist drainage and prevent premature healing.

#### *Creation of a fistula*

A fistula (Chapter XIV) is an abnormal communication between two epithelial-lined viscera or between one such viscus and the surface of the body.

In a patient with a lesion obstructing a hollow viscus (Chapter XXIII) operation is sometimes performed for the express purpose of creating a fistula. Operations of this kind are denoted by the suffix *-ostomy*; thus *gastrostomy* means making an opening into the stomach, *gastro-jejunostomy* means making a fistula between the stomach and the jejunum and *colostomy* means making an opening into the colon. Gastrostomy may be performed for the purpose of feeding a patient with obstruction of the oesophagus; gastro-jejunostomy is performed for the relief of pyloric obstruction; and colostomy may be necessary to permit passage of faeces in patients with obstruction of the colon or rectum.

*Removal of diseased or abnormal structures*

Operations are commonly undertaken for the removal of a diseased or injured structure such as a carious tooth which is beyond the stage at which it can be treated conservatively, a gangrenous leg, a pyonephrotic kidney or a part of the colon affected by carcinoma; or an abnormal formation such as a calculus in the submaxillary duct or the ureter.

When a whole organ or tumour is removed the term *excision* is used or the suffix -ectomy is appended to the name of the organ; we thus speak of excision of the appendix or appendicectomy; excision of the kidney or nephrectomy. When only a portion of an organ is removed the term *resection* is used. When a cavity is opened to gain access to some structure inside the suffix -otomy is used; thus craniotomy means opening the skull, laparotomy means opening the peritoneal cavity.

*Interruption or excision of normal structures*

At first sight it would appear quite irrational to divide or excise a normal structure but it is sometimes necessary, especially in neuro-surgery. Three examples will suffice:

1. The sensory root of the Gasserian ganglion is often divided in patients with trigeminal neuralgia (Chapter XXI). The root itself is quite normal but it transmits abnormal pain impulses.
2. The auriculo-temporal nerve is divided to stop secretion by the parotid gland in some cases of salivary fistula.
3. Part of the ganglionated sympathetic chain in the lumbar region is sometimes removed in patients with obliterative arterial disease (Chapter XVII) of the lower extremities. This operation, which is termed *sympathectomy*, causes vasodilation, especially of small vessels supplying the skin and subcutaneous tissues.
4. Castration is sometimes performed to retard the progress of a carcinoma of the prostate.

*Operations for the arrest of haemorrhage*

The ideal operation is to find and ligate the bleeding point. This is not always practicable, and it may therefore be necessary to ligate one or more vessels at some distance from the site of bleeding; the external carotid artery, for instance, is sometimes ligated to control haemorrhage after tonsillectomy when other methods have failed. Alternatively, it may be necessary to excise a viscus; splenectomy, for example, is usually required to control bleeding due to a severe injury of the spleen. The matter is discussed further in Chapter VIII.

*Operations to provide local rest to a diseased or injured part*

Operations of this type have been used extensively in the treatment of tuberculous lesions (Chapter XIII).

In tuberculous arthritis the operation of *arthrodesis* (Chapter XX) is sometimes performed. This permanently abolishes all movement at the joint so that complete rest is obtained.

In pulmonary tuberculosis before the development of effective chemotherapy (Chapter XIII) it was commonplace to try to put the whole or part of a diseased lung at rest by introducing air into the pleural cavity (*artificial pneumothorax*), by crushing or dividing the phrenic nerve to paralyse the diaphragm, or by resecting ribs in order to allow the chest wall on the affected side to cave in and thus bring about permanent collapse of the lung (*thoracoplasty*). Today, however, pneumothorax and phrenic crush are obsolete, and thoracoplasty is used mainly to reduce the volume of the thoracic cavity when a considerable amount of lung tissue has been destroyed by disease or resected.

*Reconstructive operations*

Reconstructive operations are of many kinds and range from simple wound suture or the untwisting of a volvulus to the most complex of plastic surgical procedures.

Ideally the object of a reconstructive operation is to restore normal structure and function, but this is often impossible and the best available compromise must then be sought.

When tissue is missing the deficiency can sometimes be made good by using healthy tissue in the vicinity of the lesion, as in the repair of hare lip (Chapter III). In other cases tissue is brought from elsewhere in the patient's body (*autotransplantation*), from another person (*homotransplantation*) or occasionally from an animal (*heterotransplantation*). Generally speaking autotransplants survive permanently if once they become established whereas homotransplants and heterotransplants are rejected. There are, however, some important exceptions to this rule, and homotransplants of some tissues such as bone, even when they do not survive, may provide a useful temporary scaffolding which facilitates repair by the patient's own tissue.

**Preparation of the patient for operation**

Operations often have to be undertaken as emergency procedures, but whenever possible a patient should be admitted at least 24 hours before major surgery is undertaken.



A detailed history is taken and the patient is asked about recent illnesses, drug idiosyncrasies and any tendency to abnormal bleeding; after this a thorough clinical examination is made and the urine is tested for sugar and albumen. It is then possible to form a reasonable judgment as to whether operation is justified and what risk is entailed, and to plan the operation and decide on the best type of anaesthetic.

### *General methods of preparation*

Medical treatment, which may include blood transfusion if the patient is anaemic, and dental treatment to eliminate oral sepsis, are given as required.

The patient has a bath unless he is too ill. He is then put to bed and given a light diet. It is usual to administer an enema, but drastic purgation should be avoided.

If the patient is used to alcohol it is best to allow some, but smoking should be curtailed or forbidden. A sedative is given the night before operation.

If the operation is timed for 10 a.m. the patient may have a cup of tea at 6 a.m. but nothing to eat. Pre-operative medication (typically atropine one-hundredth of a grain and morphine one-sixth of a grain, by sub-cutaneous injection) is given at 9 a.m. The appropriate skin area is washed and shaved and may be painted with antiseptic solution. Before leaving the ward the patient is told to pass urine or if necessary is catheterized.

If dentures are worn they must be removed before an anaesthetic is given, otherwise they may be inhaled or swallowed and become impacted in the pharynx, larynx or oesophagus (Chapter XXIII). In hospital it is the duty of the ward staff to remove dentures before the patient is sent to the theatre and the responsibility of the anaesthetist to check that this has been done.

### *Special pre-operative treatment*

Special pre-operative treatment may be required if the patient is suffering from shock, diabetes, severe malnutrition, thyrotoxicosis (Chapter XXII) or other disorders.

A diabetic patient should be properly stabilized—that is, the appropriate diet and insulin dosage determined—in all but the most urgent cases. Even when this has been done diabetics are liable to become acidotic following operation, especially operations performed under general anaesthesia; to prevent this an injection of insulin (20 units in an average case) should be given pre-operatively together with glucose by mouth (2N grams where N = number of units of insulin given).

### **Theatre ritual and technique**

In modern surgery an elaborate theatre ritual is practised to minimize the risk of sepsis.

Everyone in the theatre wears a mask and either clean white clothes or a theatre gown. The mask is to prevent droplet infection from the nose and throat (Chapter X). To be effective the mask must contain a sheet of some impermeable material like cellophane; in addition, coughing and sneezing must be avoided and talking reduced to a minimum.

The surgeon and his assistants and the theatre sister spend at least 10 minutes 'scrubbing up' and then put on sterile gowns and rubber gloves. All instruments and sutures are sterile and are set out on trays covered with sterile towels.

The patient is usually anaesthetized in an ante-room and then wheeled into the theatre and lifted on to the table. The skin in the vicinity of the proposed incision is cleaned and painted with antiseptic solution, and sterile sheets and towels are placed so that only the field of operation is exposed. Sterilization of a cavity like the mouth is impossible, but the risk of serious infection after major operations in this area can be greatly reduced by preliminary attention to septic teeth and tonsils and by administration of antibiotics (Chapter XI).

*The operation is planned in the light of the anatomy of the part.* Exposure must be adequate, but designed so that the risk of damage to important structures—especially nerves—is minimal. Where possible the skin incision is made in a natural crease line, and in the deeper dissection natural cleavage planes are sought and followed. Important structures are identified and gently held out of the way.

*Every care must be taken to avoid infection.* Some precautions have already been described. In addition, the following measures are adopted:

1. Small towels are clipped to the wound edges as soon as the incision has been made, so that none of the patient's skin is exposed. Even though the skin has been cleansed and painted with an antiseptic it is not absolutely sterile; it must therefore not be allowed to come into contact with instruments or gloves.

2. The hand is introduced into the wound as little as possible, and in some cases—notably orthopaedic operations—a strict 'no-touch technique' is used in which all dissection and tying of ligatures is done with instruments.

3. Sometimes antibiotics (Chapter XI) are introduced locally before the wound is closed.

*Haemostasis must be scrupulous.* Even with care the amount of blood lost during a major procedure like excision of the rectum for cancer may exceed a litre. In such cases transfusion in the theatre may be required, but even when this is given it is important to keep blood loss to a minimum. Haemostasis is also important because even a small collection of blood in a wound delays healing and predisposes towards infection.

*Gentleness in handling tissues is important.* It reduces operative shock and makes for rapid healing and recovery.

*Deep structures must be repaired and the wound closed carefully in layers.* Either catgut or a non-absorbable material such as linen, silk or steel wire is used for the muscles and fascia; and linen, silkworm gut or nylon is used for the skin.

Sometimes drainage must be provided. After evacuating pus from an acute abscess the whole wound should be left widely open, but in other cases it is usually sufficient to insert a rubber drain and close the wound around it.

At the end of the operation the wound is covered with a dry sterile dressing as a further protection against infection.

Rapidity is less important than it used to be because of improvements in anaesthesia and the development of blood transfusion, *but an operation should be performed as quickly as possible without neglecting the foregoing principles.*

### **Post-operative care**

Post-operative treatment begins in the theatre as soon as the operation is finished.

The patient is lifted *gently* on to a trolley and kept warm with blankets. A *free airway* is maintained and he is accompanied back to the ward by a nurse. There he is put into a warmed bed with his head turned to one side. When he begins to stir he may be propped up and given an injection of *morphia* unless he has an idiosyncrasy to this substance when some other analgesic drug must be used instead.

An intravenous infusion, e.g. 5 per cent dextrose or 0.9 per cent NaCl, or a blood transfusion is given in appropriate cases. The time of starting fluids or food by mouth depends on the nature of the operation.

In clean cases the dressings are usually left undisturbed until the skin sutures are due to be removed (usually 5 to 12 days after operation), but if there is much discharge daily or even twice-daily dressings may be required. These, like the operation, are carried out with aseptic precautions. All concerned wear masks, wash their hands and handle dressings with instruments only.

During the post-operative period the patient's temperature, pulse rate and respiration rate are recorded (twice daily, 4-hourly or even more frequently in special cases) and a record is kept of when he vomits, when his bowels move and how much urine he passes each 24 hours.

These procedures are routine. Additional special treatment may be required to deal with post-operative complications. In this chapter we shall consider only the complications of operations in general; the special complications of certain particular operations will be discussed later in the book.

### **Complications of operations in general**

The complications of operations in general will be considered under the following headings:

1. Shock.
2. Pain and restlessness.
3. Vomiting.
4. Dehydration.
5. Acidosis and alkalosis.
5. Retention of urine.
7. Haemorrhage.
8. Sepsis.
9. Acute parotitis.
10. Bedsores.
11. Venous thrombosis.
12. Chest complications.

#### *Shock*

Shock is liable to follow any serious operation. The prophylaxis and treatment are discussed in Chapter VIII.

#### *Pain and restlessness*

Pain and restlessness are grouped together because severe restlessness following operation is nearly always due to pain, except when the patient is coming round from the anaesthetic.

If no unusual cause for the pain can be found an analgesic drug should be given. Morphia is the best drug to use unless there is some definite contra-indication such as idionsycrasy.

#### *Vomiting*

Vomiting often occurs in the first 24 hours after operation though with modern anaesthesia it is rarely severe. If vomiting persists the cause must

be sought and the patient's stomach kept empty by means of a small tube, which is usually passed via the nose. In such cases fluid must be given *parenterally*; that is, by some route other than the alimentary canal. Most commonly it is given intravenously.

### *Dehydration*

Dehydration is common after operations because fluid loss is often great as a result of exposure of tissues, sweating, vomiting and haemorrhage, and intake is restricted. In severe cases intravenous infusion is indicated.

### *Acidosis and alkalosis*

To understand the meaning of the terms *acidosis* and *alkalosis* it is necessary to know how the acid-base equilibrium of the body is maintained. The following brief discussion is intended to remind the reader of some facts of surgical importance; for a full account of the subject and an explanation of the terms 'pH' and 'buffer' a textbook of physiology should be consulted.

The pH of the blood must not vary except within narrow limits, otherwise death occurs. Maintenance of the normal pH depends on:

1. The buffering action of the blood.
2. Elimination of carbon dioxide by the lungs.
3. Proper functioning of the kidneys.

The buffering action of the blood is due partly to the plasma and partly to the red blood corpuscles; for our present purpose, however, it will suffice to consider only the plasma.

The buffering action of the plasma is due mainly to the presence of sodium bicarbonate; this substance constitutes what is termed the *alkali reserve*. If acidic substances are administered the alkali reserve becomes decreased and the patient is then said to be in a state of acidosis. The same thing happens if excessive amounts of acidic substances are formed in the body; this may occur in diabetics, or in normal people as a result of starvation. If, on the other hand, large quantities of alkali are administered or hydrochloric acid is lost from the stomach by vomiting, the alkali reserve becomes increased and the patient is said to be in a state of alkalosis.

In moderate acidosis or alkalosis the regulating mechanisms are effective, and the pH of the plasma remains within normal limits; in extreme cases these mechanisms fail and death results.

Acidosis sometimes follows severe operations as a result of the prolonged starvation which the patient may have to undergo or because of impaired renal function. The treatment is to correct any associated fluid or electrolyte deficiency by the intravenous infusion of glucose solution or saline, and in addition to give alkali. In mild cases sodium bicarbonate may be

given orally; in more severe cases sodium lactate or sodium bicarbonate is given intravenously.

Alkalosis may develop if post-operative vomiting is severe or if a tube is used to keep the stomach empty for a long time. If the condition is suspected a sample of blood is sent to the laboratory for determination of the alkali reserve. Control of the vomiting and replacement of chloride in the form of intravenous saline are the essentials of treatment.

### *Retention of urine*

Retention of urine may follow any operation, but is especially common after abdominal and perineal operations. Male patients often find it easier to pass urine if allowed to get up and sit on the edge of the bed; if this fails the patient may have to be catheterized.

### *Haemorrhage*

Either reactionary or secondary haemorrhage may occur. The meaning of these terms and the treatment of haemorrhage are described in Chapter VIII.

### *Sepsis*

Bacterial contamination of the field of operation may occur from outside or from within the body.

Contamination from without may occur in any operation. Precautions to prevent it have already been described; if these are neglected infection may be introduced on hands, instruments or ligatures, or by droplet infection.

Contamination from within occurs only in special cases; it is most common in operations on the alimentary tract.

The results of bacterial contamination depend on the number and virulence of the organisms and the resistance of the patient. These matters will be discussed in subsequent chapters; for the moment it is sufficient to note that the local resistance is likely to be diminished if haemostasis has not been adequate.

Treatment of an infected wound is on the lines described in Chapter X. The essentials are provision of free drainage for pus by removal of sutures or other means, and chemotherapy.

### *Acute Parotitis*

Acute parotitis was once a common post-operative complication. It is less common nowadays because more attention is paid to oral hygiene but is

still occasionally seen in debilitated patients, especially those with gross dental sepsis.

Infection reaches the parotid gland from the mouth via Stenson's duct.

### *Bedsore*

A bedsore is a form of *gangrene* (Chapter XVI) due to local pressure. Bedsore occurs usually in elderly or debilitated patients who have been kept in bed for a long time, and in patients suffering from paraplegia (Chapter XXI).

Prophylaxis is all-important and consists in reducing the pressure over bony prominences by ring cushions, altering the position of the patient every hour or two and applying spirit followed by a dusting powder to areas where sores threaten to develop.

When a bedsore has occurred it is covered with a dry sterile dressing; alternatively, an occlusive elastoplast dressing is applied. The patient is nursed in such a position that the sore is not subjected to pressure. As a rule the slough separates after some weeks and the area then slowly heals. Sometimes, however, sloughs have to be excised and plastic operations may be required to expedite the healing of large defects.

### *Venous thrombosis and pulmonary embolism*

Thrombosis (Chapter XVII) in the veins of the calf, the femoral vein or the veins of the pelvis is a common complication, especially of abdominal operations. If the leg veins are involved the patient complains of pain and the limb becomes swollen, tender, and sometimes oedematous. The chart usually shows an evening temperature of 99°–100° F.

The danger of the condition is that the whole or part of the thrombus may become detached and travel via the vena cava, right auricle, tricuspid valve and right ventricle to the pulmonary artery. This complication, which is known as pulmonary embolism, occurs most commonly in the second week after operation.

Blockage of the trunk of the pulmonary artery or one of its two main divisions causes sudden severe pain in the chest, marked dyspnoea and cyanosis. Death may occur within a few minutes. If one of the smaller branches is blocked the symptoms are less severe and the patient usually recovers. His sputum often becomes blood-stained within a day or two, and examination of the chest may reveal an area of consolidation.

*Treatment.* As a prophylactic measure the patient is told to move his limbs freely in bed and under no circumstances is a bolster placed under his knees. He is made to get up as soon as possible after operation.

If thrombosis develops in the leg veins the limb is elevated and, provided there is no contraindication, anticoagulant drugs (*heparin* or *phenindione*) are administered. If the thrombus appears to be spreading the femoral vein may be ligated in the thigh to combat the danger of embolism.

Should pulmonary embolism occur the patient is given morphia by injection and oxygen through a mask. If he recovers injections of penicillin are given for a few days to combat the risk of infection in the damaged area of lung. A few lives have been saved by the operation of pulmonary embolectomy. This procedure, which was first attempted by Trendelenberg in 1907, has become much more feasible in recent years with the development of methods of cardio-pulmonary bypass.

#### *Other chest complications*

Chest complications may follow any major operation, whether performed under local or general anaesthesia. In addition to pulmonary embolism they include:

1. Bronchitis.
2. Pulmonary collapse.
3. Pneumonia.
4. Lung abscess.
5. Gangrene of the lung.
6. Pleural effusion and empyema.

We shall describe these conditions and discuss their etiology, prevention and treatment. Diagnosis will not be considered in detail since it depends largely on physical and radiological examination of the chest. These procedures are described in textbooks of medicine, but are beyond the scope of the present book.

*Bronchitis*—that is, inflammation of the bronchi—is a common post-operative complication. It usually resolves completely, but may lead to pulmonary collapse or bronchopneumonia. The main symptoms are fever and cough with the production of purulent sputum.

*Pulmonary collapse*, sometimes called *atelectasis*, occurs in two forms: *lobular* collapse, affecting multiple small areas of lung; and *lobar* collapse, affecting the whole of one or more lobes. In both forms the pulmonary alveoli in the affected areas cease to contain air. The cause of pulmonary collapse has been much disputed but is now known to be bronchial obstruction, usually due to adherent mucus. This prevents entry of air, and air distal to the obstruction is absorbed into the blood stream.

The symptoms and signs of collapse usually appear within 48 hours of operation, but may be delayed until the fourth or fifth day. In lobular collapse there is a moderate increase in the temperature and pulse rate,



and the patient has a cough which may or may not be productive of sputum. In lobar collapse the patient becomes suddenly dyspnoeic and cyanosed, and his temperature rises; the physical signs are those of consolidation in the affected lobe and shift of the mediastinum towards the affected side.

Infection is likely to supervene in the collapsed areas. This usually results in pneumonia, but occasionally, especially in debilitated patients, abscess or gangrene of the lung develops.

*Pneumonia* is acute inflammation of the lung with consolidation. There are two main forms, *bronchopneumonia* and *lobar pneumonia*, either of which may occur as a complication of operation. In bronchopneumonia the consolidation is patchy, and there is a mixed infection with streptococci usually predominating; in lobar pneumonia a large area of lung, often comprising one or more lobes, is affected, and the causal organism is the pneumococcus. A particularly dangerous type of bronchopneumonia is sometimes caused by inhalation of vomitus, pus or other foreign material.

In pneumonia fever is high, the pulse and respiration are rapid, and the patient is often cyanosed. The sputum is purulent and may be tinged with blood. The main differential diagnosis is from pulmonary collapse.

*Lung abscess.* An abscess is a circumscribed collection of pus (Chapter X). Lung abscess may develop as a complication of collapse or pneumonia, but is most often due to inhalation of foreign material. Inhalation of vomitus and pus has already been mentioned as a cause of septic bronchopneumonia; it may also cause lung abscess. In addition, abscess may result from inhalation of a solid foreign body; for example, a tooth or tooth-fragment.

The development of a lung abscess is usually marked by the appearance of a high swinging temperature and an increase in the pulse and respiration rates. Sweating and bouts of uncontrollable shivering termed rigors are common. The abscess commonly ruptures to a bronchus; following this the fever may abate and the patient expectorates large amounts of foul pus and sometimes blood.

X-ray examination is important since in addition to confirming the presence of an abscess it may reveal an opaque foreign body.

*Gangrene of the lung.* In debilitated patients and diabetics instead of abscess formation a portion of lung may undergo necrosis and putrefaction. The breath and sputum become extremely offensive and the patient appears desperately ill. This condition, which is termed gangrene of the lung, is usually fatal.

*Pleural effusion and empyema.* After some operations, especially on the upper abdomen, an effusion of clear fluid develops in the pleural cavity. This fluid may become purulent owing to super-added infection and the

condition is then known as empyema. In other cases empyema develops as a complication of pneumonia or lung abscess.

The diagnosis of empyema is suggested by the presence of a high swinging temperature in association with signs of fluid on physical examination of the chest. It is confirmed by aspirating a little fluid through a needle inserted through one of the intercostal spaces.

*Prevention of post-operative pulmonary complications.* The incidence of pulmonary complications can be greatly reduced by taking precautions before, during and after operation.

All but the most urgent operations should be postponed if the patient has a cold or other respiratory infection. If operation is unavoidable local or spinal anaesthesia should be used if possible; failing this nitrous oxide and oxygen, or cyclopropane, is usually best. Ether should not be used. Pre-operative injection of atropine is especially important in these cases.

The patient's stomach should be empty when he goes to the theatre. If he is vomiting a tube should be passed to empty the stomach and left in place.

During operation a free airway must be maintained and the patient should never become cyanosed. Anaesthesia should not be deeper than the surgeon requires.

Where there is special danger of inhalation of foreign material the anaesthetic should be given through an intratracheal tube. One with an inflatable cuff should be used or else gauze should be packed around the tube in the pharynx.

After operation the patient should be made to cough and do breathing exercises, if possible under the supervision of a physiotherapist. His position should be changed every hour.

Post-operative sedation requires careful consideration. If sedatives and analgesics are withheld the patient may be afraid to cough on account of pain; on the other hand, if large doses are given the patient is too drowsy to cough voluntarily and reflex coughing is impaired.

*Treatment.* There are two essentials in the treatment of pulmonary collapse; the first is to get rid of the mucus or muco-purulent exudate in the bronchi; the second is to prevent the development of infection in the collapsed areas.

Elimination of bronchial exudate depends primarily on the patient's own efforts; he *must* be convinced of this and persuaded to cough. In addition, drainage should be encouraged by frequent changes of posture and, if possible, by making the patient lie for part of the time on his sound side

with the foot of the bed raised. In massive collapse these measures may not be sufficient and an attempt may then be made to remove the mucus plug through a bronchoscope. Very occasionally tracheostomy is required.

Antibiotics are given to prevent infection. The choice of drug, means of administration and dosage are discussed in Chapter XI.

Bronchitis and pneumonia are also treated by encouraging coughing and giving appropriate antibiotics. If the patient is cyanosed oxygen should be administered through a mask.

The treatment of lung abscess and empyema is beyond the scope of this book.

### Chapter III

## CONGENITAL DISORDERS

A disorder, whether in the form of a general disease or a localized lesion, is described as congenital if it is present at birth. Many such disorders are obvious at once; sometimes, however, though the underlying defect is present at birth, symptoms do not appear until later.

From an etiological point of view congenital disorders may be subdivided into those due to an abnormality present in the fertilized ovum, and those due to intra-uterine disease. Abnormalities in the fertilized ovum might conceivably result from the process of *mutation*; usually, however, they are due to the presence of particular *genes* derived from one or both parents, and the resulting disorders are then spoken of as *hereditary*. Intra-uterine disease is obviously the cause of congenital syphilis, but has, until recently, been regarded as a very rare cause of other congenital disorders. It has been found, however, that some congenital deformities occur as a result of the mother contracting *rubella* ('German measles') during pregnancy, and it seems possible that other virus diseases may produce similar results.

From a theoretical point of view the etiological classification given above is ideal, but in practice there are many congenital disorders whose etiology is still unknown. However, even though we may not know precisely why a particular disorder occurs we can often discover something of the mechanism of its production from a study of normal embryological development; some knowledge of embryology is therefore essential for an understanding of the present discussion.

It is unnecessary to attempt to review here the whole field of congenital disorders. Instead we shall describe in detail a number of examples which are of special interest to dental students. In so doing we shall, where necessary, give a brief résumé of the relevant embryology.

### ✓ Haemophilia and other Hereditary Haemorrhagic Disorders

Haemophilia is a rare hereditary disease of males, characterized by a tendency to uncontrollable haemorrhage following slight trauma. It is due to a defective gene carried on the X chromosome resulting in a complete or partial deficiency of one of the factors necessary for blood clotting termed

Factor VIII or *antihaemophilic globulin* (Chapter VIII). The gene responsible is, in Mendelian terminology, a sex linked recessive, and the condition is therefore transmitted to male children by mothers who themselves appear perfectly normal.

The bleeding tendency is usually noticed in infancy, and it is frequently possible to obtain a definite family history. Haemorrhage may occur internally, especially into joints and subcutaneous tissues, or externally. The clotting time of the blood is greatly prolonged.

Haemophilia is of considerable surgical importance because sufferers from this disease may bleed to death after quite minor surgical procedures such as circumcision or dental extraction unless special precautions are taken (Chapter VIII).

*Christmas disease* resembles haemophilia clinically but is due to deficiency of a different clotting factor known as Factor IX (Chapter VIII).

*Von Willebrand's disease* is a hereditary disorder inherited as an autosomal dominant in which the bleeding time is prolonged but the blood as a rule clots normally. Severe bleeding may occur from small vessels. The precise nature of the defect is not known (Chapter VIII).

### **Congenital Syphilis**

A syphilitic mother can transmit the disease to her offspring long after she has ceased to be sexually contagious. If the disease is transmitted to the foetus in the early stages of the mother's disease a miscarriage usually results. If transmitted at a later stage the child may be still-born, or born alive but suffering from congenital syphilis. The manifestations of this condition are numerous and will be described later (Chapter XIII).

### **Hare Lip, Cleft Palate and other Congenital Lesions of the Face and Lips**

#### **Embryology—Development of the Face, Lips and Palate**

The face is formed from five processes which appear round the *stomodaeum* or primitive mouth about the fifth week of intra-uterine life. These are the *frontonasal process* above (growing down from the headfold), and a *maxillary* and *mandibular process* on each side below (arising from the first branchial arch). Two olfactory pits soon appear in the frontonasal process, dividing it into a *median nasal* and two *lateral nasal* processes. Between the lateral nasal and maxillary process on each side lies the naso-orbital fissure, which later becomes infolded to form the lachrymal duct. (See Fig. 9.)

The *upper lip* is formed from the two maxillary processes and the median nasal process. The maxillary processes first fuse with the median nasal process and then, according to most modern embryologists, send medial extensions which cover over the median nasal process and fuse together in the midline. On this view only the deep aspect of the median portion of the lip is formed from the median nasal process; all the rest of

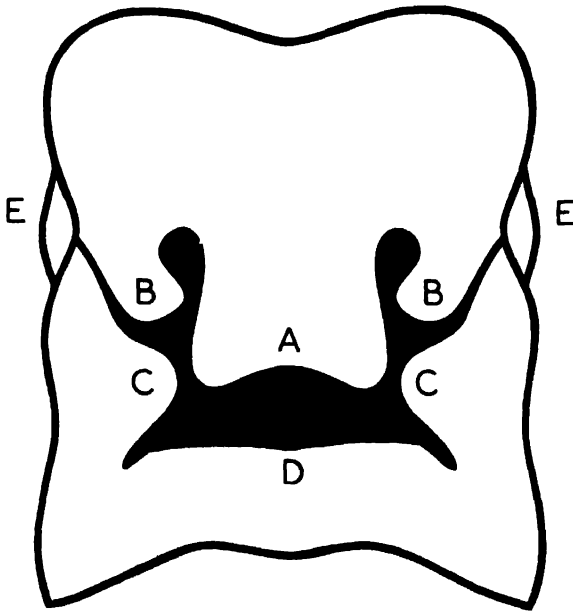


FIG. 9. Diagram illustrating the development of the face.

- |                          |                                |
|--------------------------|--------------------------------|
| A. Median nasal process. | BB. Lateral nasal processes.   |
| CC. Maxillary processes. | D. Fused mandibular processes. |
| EE. Developing eyes.     |                                |

the lip is formed from the maxillary processes. The lateral nasal processes help to form the side of the face, but play no part in the formation of the lip.

The *nasal septum* is formed by an inward prolongation from the median nasal process.

The *palate* is formed mainly by ingrowths from the two maxillary processes, which fuse in the mid-line with each other and with the developing nasal septum. The premaxillary part of the palate (that is, the part lying anterior to the incisive foramen) develops as an ingrowth from the median nasal process.



FIG. 10. Unilateral hare lip.

*A and B.* Infant with partial hare lip before and after operation.

*C and D.* Infant with complete hare lip and cleft palate before operation, and shortly after repair of the lip and the anterior part of the palate.  
(Mr W. Hynes' cases)



FIG. 11. Bilateral hare lip with prominent pre-maxilla.

- A. Before operation.
- B. Shortly after repair of the lip and anterior part of the palate on left side.
- C. After repair of the lip and anterior part of the palate on right side.

Additional plastic operations will be performed to elevate the tip of the nose and improve the appearance of the lip.  
(Mr W. Hynes' case)



**Hare lip and cleft palate**

The term *hare lip* denotes a congenital cleft in the upper lip due to failure of fusion between the median nasal process and one or both maxillary processes.

Hare lip may thus be *unilateral* or *bilateral*. It is further classified as *simple* or *alveolar*, depending on whether it involves the soft parts only or the soft parts together with the alveolus; and as *complete* or *incomplete*, depending on whether it does or does not extend into the corresponding nostril. The commonest type is the unilateral complete, and the next commonest the unilateral incomplete (Fig. 10). Bilateral clefts are fortunately relatively rare; when they occur they are usually alveolar (Fig. 11).

In every case of alveolar hare lip the palate is cleft and the defect in the alveolus is continuous with that in the palate.

The main symptom in cases of hare lip is the deformity, but in the more severe forms there may also be difficulty in suckling.

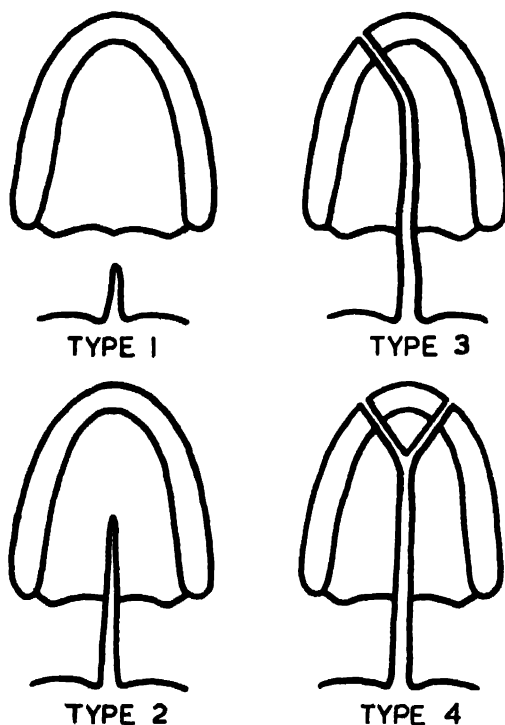


FIG. 12. Diagram illustrating the varieties of cleft palate. For explanation see text.

*Cleft palate* is also congenital and results from failure of fusion between the palatal parts of the maxillary and median nasal processes.

Four varieties of cleft occur, as illustrated in Figure 12. In Type I the uvula and soft palate alone are affected. In Type II the cleft involves the hard palate for a varying distance, but does not extend further forward than the anterior palatine foramen. In Type III there is a complete unilateral cleft of the palate and the alveolar margin, associated with a unilateral hare lip, and in Type IV there is a complete bilateral cleft with a double hare lip. It will be seen that in complete unilateral cases the cleft is in the mid-line posteriorly but anteriorly deviates to one or other side, the alveolus being split usually just lateral to the corresponding central incisor tooth; in complete bilateral cases the cleft is Y shaped, being in the mid-line posteriorly and having diverging anterior limbs.

Cleft palate may interfere seriously with suckling, speech and development of the teeth.

### *Treatment*

It is important to determine at birth the state of the alveolus. If it is cleft on one side the premaxilla is often rotated to the non-cleft side and this tends to allow the palate on the cleft side to collapse medially and so disturb the normal curvature of the alveolar arch. It is possible to correct this deformity with orthodontic plates provided that this treatment is begun soon after birth. The plates have the added advantage that feeding is assisted and most babies take the bottle well; if they do not do so spoon feeding is undertaken. In bilateral clefts of the alveolus the premaxilla is often rotated anteriorly and both lateral parts of the palate collapse medially. Again, orthodontic splints can materially improve the arch provided treatment is begun early.

Many different operations have been devised for the treatment of hare lip and cleft palate.

The current practice is to operate on the lip at the age of 3 to 4 months, provided that the baby is of normal weight and thriving. General anaesthesia is required in all cases. The principle of the operation is to mobilize the lip from the maxilla, pare the edges of the cleft and then suture mucosa, muscle and skin. In the case of alveolar clefts the anterior portion of the palate cleft is usually repaired at the same time as the lip. In bilateral cases a mass of tissue developed from the median nasal process often projects in a most unsightly manner and special methods of treatment are required to deal with this.

In some cases secondary plastic operations are required when the child is older to improve the cosmetic result.

Operations for cleft palate are designed to close the defect and at the same time provide a long and mobile soft palate which is able to shut off the naso-pharynx from the oral pharynx during speaking and swallowing. This helps the child to learn to speak more normally and reduces the risk of chronic nasopharyngitis and otitis media.

The soft palate is mobilized so that the two halves can be brought together; the edges of the cleft are pared, and repair is effected by three layers of sutures. One layer unites the mucous membrane on the nasal aspect, one unites the muscle and one unites the mucous membrane on the buccal aspect. Attempts to close the gap in the hard palate by forcing the bones together are futile and have long been abandoned. Instead the bony defect is covered above and below by sliding inwards flaps of mucous membrane from each side of the cleft, one pair of flaps is elevated from the nasal aspect of the bone and one from the buccal aspect.

With complete clefts it is convenient, though not essential, to repair the anterior part of the cleft with the lip at about 4 months of age. Repair of incomplete clefts, and of the posterior part of complete clefts, is usually undertaken when the child is about 12 months old; that is, before speaking has developed to any important degree. Some surgeons, however, prefer to repair the soft palate at the age of 6 to 9 months and postpone the repair of the hard palate until about 5 years of age.

In older patients who have not been treated in infancy it is sometimes best to undertake operative repair of the defect in the soft palate only, and supply a suitable obturator to close the defect in the hard palate.

A recent development is the use of bone grafting to repair alveolar defects. This results in a better arch, and hence in a better bite and less caries. The usual age for performing this operation is 6 to 8 years.

Sometimes defective speech occurs because the soft palate, though not cleft, is incompetent. The operation of *pharyngoplasty* may then be performed to reduce the size of the opening between the naso-pharynx and the oral pharynx.

Many people are concerned in the treatment of hare lip and cleft palate, including the surgeon, dental surgeon, orthodontist, pediatrician and speech therapist, and the outcome depends to a large extent on good team work. The patient should be followed up routinely until the age of 15 years, and longer if it seems necessary.

### **Other congenital lesions of the face and lips**

These include congenital *micro-stoma* and *macro-stoma*, due respectively to excessive or defective fusion of the maxillary and mandibular processes; and *oblique facial cleft* due to persistence of the naso-orbital fissure.

## **Branchial Cysts and Fistulae**

### **Embryology**

During the development of the neck five bars termed branchial arches appear, each bar consisting of a plate of cartilage, a mass of muscle, a nerve and an artery. The first branchial arch is known as the mandibular arch and has already been mentioned in connection with the development of the face.

Between the arches are depressions—the pharyngeal pouches—lined internally by the columnar epithelium of the pharynx and externally, by squamous epithelium.

The second arch grows rapidly, especially in a caudal direction, and so comes to overlap the third and fourth arches; finally it meets and fuses with the fifth arch. A space is thus formed which is lined with squamous epithelium and is known as the cervical sinus. If development proceeds normally the sinus becomes completely obliterated, but sometimes it persists, giving rise to a *branchial cyst*. Occasionally the second arch fails to fuse with the fifth, and a track is then formed which is lined with squamous epithelium and opens on the surface of the neck. This is known as a branchial fistula, though it should strictly be termed a branchial sinus (see Chapter XVI).



FIG. 13. Branchial cyst. The outline of the cyst and the borders of the sternomastoid muscle have been marked on the patient's skin.

### **Branchial cyst**

A branchial cyst (Fig. 13) rarely becomes apparent before puberty, and sometimes not until the patient is over twenty. The reason is that in early life the cyst is small and empty, but later it becomes distended with fluid secreted by the lining epithelium. It then forms a smooth fluctuant swelling in the lateral region of the neck at about the level of the angle of the jaw.

It lies partly deep to the sternomastoid and partly in front of the anterior border of this muscle. Branchial cysts are liable to recurrent attacks of inflammation. An uninfected cyst is not attached to the skin and is often translucent; an infected cyst may be attached to the skin and is never translucent.

The most important differential diagnosis is from tuberculous lymphadenitis (Chapter XIII). Early in this condition the enlarged lymph nodes are solid and no difficulty arises, but later liquefaction occurs and a fluctuant swelling may develop in the position of a branchial cyst. This swelling is usually attached to the skin and is never translucent; in addition, it is usually possible to feel enlarged nodes in which liquefaction has not yet occurred. The differential diagnosis can, therefore, usually be made on ordinary physical examination, though an infected cyst sometimes causes difficulty. If doubt exists some fluid should be aspirated from the swelling. Fluid from breaking down tuberculous nodes is thick and creamy, and consists of a suspension of fatty debris containing very few cells. Fluid from an infected cyst is true pus and contains numerous polymorphonuclear leucocytes; that from an uninfected cyst is glairy and viscid, and has a shimmering appearance due to the presence of numerous cholesterol crystals.

Does carcinoma ever develop in branchial epithelium? The answer is rarely, if ever. A hard fixed swelling in the lateral region of the neck is usually due to secondary carcinomatous deposits in lymph nodes (Chapter XIV). If no primary tumour can be found a diagnosis of branchogenic carcinoma is sometimes made, but in such cases it nearly always transpires that a small primary lesion in the naso-pharynx or pyriform fossa has been overlooked.

### *Treatment*

A branchial cyst should be removed by operation.

### **Branchial fistula**

A branchial fistula begins to discharge soon after birth. The opening of the fistula is situated at the anterior border of the sternomastoid muscle, usually about an inch above the sternoclavicular joint (Fig. 14). The fistula extends upwards, pierces the deep fascia and passes deeply between the external and internal carotid arteries to the region of the wall of the pharynx.

The patient suffers a certain amount of inconvenience, but operative treatment is difficult and is not always necessary. Sometimes the fistula is the site of recurring attacks of inflammation, and then operative removal should be undertaken.



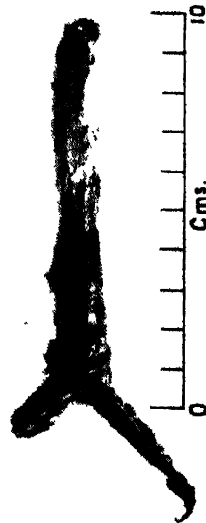
FIG. 14. Branchial fistula.

- A. The patient, showing the opening of the fistula above the right sternoclavicular joint.
- B. Radiograph taken after injection of lipiodol into the fistula.
- C. The specimen removed at operation. It includes an ellipse of skin, and tissue surrounding the fistula throughout its whole length.

(Mr Charles Donalds' case)



B



C

## **Congenital Lesions Associated with the Development of the Thyroid Gland**

### **Embryology**

The thyroid gland is developed mainly from the thyroglossal duct, which grows down into the neck in the mid-line from the region of the foramen caecum of the tongue, and partly from outgrowths from the fourth pharyngeal pouches.

The thyroglossal duct normally becomes completely obliterated but sometimes portions of it persist.

Three types of developmental anomaly may occur: thyroglossal cysts, accessory thyroids and ectopic thyroid.

### **Thyroglossal cyst**

A thyroglossal cyst (Fig. 15) may occur anywhere in the line of the thyroglossal duct; that is, anywhere in, or close to, the mid-line between the base of the tongue and the normal position of the thyroid gland. Such cysts are connected to the base of the tongue by the remains of the duct.

A thyroglossal cyst, unlike a branchial cyst, becomes apparent in childhood, usually about the age of four. It presents as a mid-line, or nearly mid-line, cystic swelling, and has the special characteristic of moving upwards when the tongue is protruded. It is not attached to the skin except as the result of recurrent inflammation.

The treatment is operative removal of the cyst and the whole of the remains of the thyroglossal duct up to the base of the tongue. Incomplete removal may be followed by the appearance of a so-called thyroglossal fistula (Fig. 17), properly termed a thyroglossal sinus. A thyroglossal fistula, unlike a branchial fistula, never occurs as a primary lesion but only as the result of rupture, incision or inadequate removal of a thyroglossal cyst.

### **Accessory thyroids**

Accessory thyroid tissue may occur anywhere along the line of the thyroglossal duct, and also in the vicinity of the lateral lobes of the thyroid gland.

There is some evidence that accessory thyroid tissue is prone to the development of carcinoma in later life, and it should therefore be removed. Before this is done, however, it is necessary to ascertain by careful palpation that there is a thyroid gland present in the normal situation.

### **Ectopic thyroid**

The term ectopic thyroid is used when there is thyroid tissue in an abnormal site, but none in the normal site. In such cases operative removal is



FIG. 15



FIG. 16



FIG. 17

FIGS. 15 and 16. These patients were both referred with a provisional diagnosis of thyroglossal cyst. In Fig. 15 the swelling is in the mid-line of the neck and moves upward when the tongue is protruded. In Fig. 16 the swelling is an inch to the left of the mid-line and does not move when the tongue is protruded.

*Diagnosis:* Fig. 15, thyroglossal cyst; Fig. 16, sebaceous cyst.

FIG. 17. Thyroglossal fistula. The fistula developed in childhood after an attack of inflammation in a thyroglossal cyst.



contraindicated because it would leave the patient bereft of all thyroid tissue.

### **Congenital Sequestration Dermoid Cysts**

Along the lines of embryological fusion small portions of ectoderm may become separated and buried beneath the surface; these may then give rise to cysts lined with squamous epithelium.

Such cysts are known as congenital sequestration dermoids. They occur only in the region of the embryological fusion lines and are filled with sebaceous material secreted by the lining epithelium. They sometimes contain hair.

Sequestration dermoids may occur in the mid-line in the floor of the mouth (sublingual dermoid), below the chin (submental or suprahyoid dermoid) and above the sternal notch (supra-sternal dermoid); and at the outer angle of the orbit (external angular dermoid—Fig. 18).

Operative removal is usually a simple matter and is often requested by the patient for cosmetic reasons.



FIG. 18. External angular dermoid.

### **Haemangiomata and Lymphangiomata**

Haemangiomata and lymphangiomata are described in Chapters XVII and XVIII respectively. The terms strictly mean tumours of blood and lymphatic vessels, but the lesions to which they refer are for the most part developmental anomalies and not true tumours.

## *Chapter IV*

# INFLAMMATION AND REPAIR

The processes of inflammation and repair are so intimately related that it is impossible to draw a hard and fast line between them. In this chapter we shall consider primarily the clinical aspects of these subjects; for a discussion of the morbid histological changes the student should consult a text-book of pathology.

No entirely adequate definition of inflammation has ever been formulated, but that of Burdon Sanderson (1870) is as good as any. 'Inflammation', said Sanderson, 'is the succession of changes which occur in a living tissue when it is injured, providing the injury is not of such a degree as at once to destroy its structure and vitality'.

Injury may result from many causes; namely:

1. Physical causes, including mechanical, thermal, electrical and other physical forms of energy.
2. Chemical causes; for example, strong acids or alkalis.
3. Microbiological causes, including bacteria, filtrable viruses, fungi and protozoa.

### **Aseptic Inflammation**

Inflammation due to physical and chemical causes, in which bacteria and other micro-organisms play no part, is called *aseptic inflammation*.

Inflammation which is initially aseptic is often complicated by subsequent bacterial infection. An important example of inflammation which remains aseptic—or very nearly so—is seen in the healing of a clean incised wound. This is called 'healing by first intention'.

The sequence of events in aseptic inflammation, and the ultimate result, depend on the tissues involved. In man, as distinct from certain more primitive organisms, the capacity for *regeneration* (restoration of the *status quo ante* following destruction of tissue) is limited to the following tissues:

1. *The Blood*. Blood is a tissue and following hæmorrhage, if the patient survives, the plasma and cells which have been lost are gradually replaced.
2. *Connective tissue*. If connective tissue is destroyed it is replaced by newly-formed connective tissue.

3. *Bone.* The union of a fracture is a familiar instance of bone regeneration.

If the periosteum is preserved normal anatomy may be restored even after the loss of quite large amounts of bone, especially in young subjects.

4. *Epithelium of simple type.* After loss of skin the epithelium may be replaced by new epithelium growing in from the margins of the lesion or from islands of living epithelial tissue which have escaped destruction. This new epithelium is, however, different in quality from that of normal skin, and if complex structures such as hair follicles have been destroyed they are not regenerated. In lesions of mucous membranes the lost epithelium may be replaced in a similar manner but again complex glandular structures are not re-formed. If organs like the liver and kidney are partly destroyed there may be proliferation of both epithelium and connective tissue but the normal structure is never completely restored.

5. *The axon fibres which make up the peripheral nerves.* After injury of a nerve the axon fibres regenerate but, for reasons which will be discussed in Chapter XXI, there is usually only partial restoration of function.

Blood clot in the tissues may be absorbed but is often invaded and replaced by connective tissue. This process is known as *organization*. Muscle, if destroyed, is not repaired by the formation of new muscle cells but is replaced by connective tissue; similarly, if the nervous tissue in an area of the central nervous system is destroyed it is replaced by *neuroglia*. Areas of connective tissue or neuroglia replacing more specialized tissues which have been destroyed are termed *scars*.

## **Inflammation due to Micro-Organisms**

### **Classification**

In clinical practice the term inflammation is often used in the restricted sense of inflammation due to micro-organisms. This custom leads to a certain amount of confusion, but as it is well established and also convenient we shall adhere to it throughout the remainder of this chapter.

Bacteria which cause injury—and hence inflammation—are termed *pathogenic*. The entrance of pathogenic bacteria into the tissues is known as *infection*.

Bacterial inflammation is commonly classified as *acute*, *subacute* or *chronic*. 'Acute' and 'chronic' are pathological terms and indicate the time relations of the process; 'subacute' is a term used by clinicians rather than pathologists, and means simply 'mild acute'.

Inflammation may also be classified as *suppurative* or *non-suppurative*, according to whether or not pus is formed, and either type may be acute, subacute or chronic. Suppurative inflammation is discussed in Chapter X. Non-suppurative inflammation of a mucous membrane is called *catarrhal inflammation*, except in the special case where there is an outpouring of fibrin leading to the formation of a so-called *false membrane*, when it is known as *croupous inflammation*. The *common cold* is an example of catarrhal inflammation due to a filtrable virus. Non-suppurative inflammation of glandular tissue, also due to a virus, is exemplified by *mumps*. Croupous inflammation is characteristic of diphtheria (Chapter XII).

Another classification of inflammation sometimes used by clinicians is into *specific* and *non-specific*. The term 'specific' is used when the clinical findings point, *or are supposed to point*, unequivocally to one particular organism, like the *spirochaete* of syphilis or the *B.anthraxis*. The term 'non-specific' is used to indicate that the condition could be due to any one of a large number of organisms; it is applied, for instance, to acute cellulitis, which may be due to different varieties of haemolytic streptococci and occasionally to staphylococci.

The student should beware of these words. In the first place many allegedly specific infections may be due to one of several organisms. Lobar pneumonia, for instance, may be caused by any one of a large number of types of pneumococcus, and tuberculosis in man may be due to the 'human' bacillus or the 'bovine' bacillus. Secondly, in using the designation 'non-specific' we are apt to forget that in *every* patient with an inflammatory lesion the cause is perfectly definite and should be determined whenever possible.

### Defences of the body against bacterial infection

Bacteria abound in the air we breathe and the food we eat; they infest our skin and are present in large numbers throughout almost the whole of our alimentary canal. While many of these organisms are harmless, many others are pathogenic; we are therefore constantly exposed to the hazard of infection. From time to time this hazard is increased; for instance, by contact with a patient suffering from an infectious disease.

The power of resisting infection—without which human life would not be possible—is termed '*immunity*'. Immunity is partly *innate* and partly *acquired*. Innate immunity is independent of present or past infection. It may be impaired or abolished by cold, loss of blood, debilitating diseases and starvation, or by local factors such as trauma or the presence of foreign bodies. Acquired immunity may develop gradually during the

course of an infection or may be due to previous infection. It may also be produced artificially by injection of bacteria, bacterial products or serum.

### *The mechanism of immunity*

Intact skin and mucous membrane provide a considerable barrier to the entrance of bacteria, but they may be rendered ineffective by quite minor injuries. If organisms gain entrance to the body they encounter two types of defence, termed respectively *cellular* and *humoral*.

*Cellular defence* is the function of cells known as *phagocytes* which have the power of ingesting and destroying bacteria. Phagocytes are of two main kinds: the polymorphonuclear leucocytes of the blood (sometimes termed *microphages*) and the *macrophages*. Macrophages are formed in various parts of the body including the spleen, lymph nodes, bone marrow, liver and, to some extent, ordinary connective tissue; in addition some macrophages are probably formed from the monocytes of the blood.

*Humoral defence* depends on the presence in the blood and tissues of substances which destroy bacteria or neutralize their toxins. These substances are known as *antibodies*; they are of various kinds: agglutinins, bacteriolysins, antitoxins and so on.

## **Clinical manifestations**

The clinical manifestations of inflammation may be discussed under two headings: local manifestations and general manifestations.

### *Local manifestations*

The classical description of the local manifestations of inflammation was given by Celsus (A.D. 50), who enumerated the four cardinal features of *pain, heat, redness* and *swelling*. To these may be added a fifth; namely, *loss of function*.

Some of these manifestations may be specially prominent, some absent; the features in any given case depend on the tissue involved, the nature and number of the invading organisms and the resistance of the patient. It will, however, be convenient to postpone further consideration of this subject until later chapters when we shall discuss various examples of bacterial inflammation in detail.

### *General manifestations*

The commonest general manifestations of inflammation are fever, increased pulse rate, loss of appetite, a dry furred tongue and constipation. After a time the lips and teeth may become covered with foul brownish material known as *sordes*, compounded of food, epithelial cells, inspissated mucus

and micro-organisms. In severe cases there may be rigors, delirium, loss of weight and, finally, exhaustion and collapse.

These manifestations are caused by bacterial toxins or actual bacteria entering the circulation. The extent to which this occurs depends largely on the nature and number of the invading organisms and the resistance of the patient, but purely anatomical factors are sometimes important. A lung abscess, for instance, may burst into a bronchus, with consequent cessation of toxin absorption; on the other hand, inflammation in the middle ear may spread to the sigmoid venous sinus and cause severe toxæmia or even septicaemia (Chapter X).

### **The course of bacterial infection**

The course of bacterial infection depends on the progress of the battle between the invading organisms and the defences of the body. We may distinguish three possibilities:

1. *The infection remains localized and the bacteria are all destroyed within a week or two.*

The most favourable result is *resolution*; that is, complete restoration of the normal structure of the part. If tissue incapable of regeneration has been destroyed complete resolution is impossible and the best that can be achieved, after the infection has been overcome, is replacement of the dead tissue by newly-formed connective tissue. This is called *healing by fibrosis*.

Some organisms cause *suppuration* (pus formation). This is of great surgical importance and will be discussed in detail later (Chapter X). For the present it is sufficient to note that *after the pus has been evacuated* resolution or healing by fibrosis may occur.

2. *The infection remains localized but persists for weeks or months.*

Symptoms like pain, redness and fever, which are particularly characteristic of acute inflammation, become less marked or disappear; swelling and loss of function, however, usually remain. This state of affairs is termed *chronic inflammation*. It may be interrupted from time to time by the temporary recrudescence of acute symptoms.

3. *Infection spreads to other parts of the body.*

This may occur by the following routes:

(1) *Spread by direct continuity.* Spread of infection by direct continuity is typical of acute cellulitis, but may occur in many inflammatory conditions. The process is analogous to the spread of a fire through a forest.

Some structures, especially those made of dense fibrous connective tissue, are resistant to the spread of infection. This explains why, in the hand, suppuration may remain for a long time localized in a tendon sheath

or in one of the fascial compartments of the palm; and why, in acute parotitis, infection rarely spreads through the parotid capsule to other structures in the neck.

(2) *Spread by lymphatics.* Infection spreading along lymphatics may or may not lead to inflammation of the vessel wall (lymphangitis), but it always leads to inflammation of the related lymph nodes (lymphadenitis). It is therefore important to know which regions of the body drain to particular groups of nodes.

Lymphangitis and lymphadenitis are described in Chapter X.

(3) *Spread by serous cavities.* A common instance is the spread of infection from an inflamed appendix throughout the peritoneal cavity (*peritonitis*).

(4) *Spread by epithelial-lined tubes and cavities.* This is exemplified by spread of infection from the nose to the accessory nasal sinuses, and from the kidney via the ureter to the bladder.

(5) *Spread within the central nervous system, especially via the subarachnoid space and ventricular system.*

(6) *Spread by the blood stream.* Infection may spread by direct continuity from a local inflammatory lesion to the wall of a vein. This is followed by clot formation (*thrombosis*). The clot becomes infected and extends along the lumen of the vein; later small pieces may become detached and swept away in the blood stream (*septic embolism*). Examples of this form of spread are: (a) from a carbuncle of the upper lip via the angular vein to the cavernous sinus; (b) from the sigmoid sinus (itself infected by direct spread from the middle ear and mastoid antrum) down the internal jugular vein; (c) from an acutely inflamed appendix via the ileocolic, superior mesenteric and portal veins to the liver.

Widespread dissemination of infection via the arterial system may also occur, as in septicæmia and pyæmia.

Thrombosis and embolism are discussed in Chapter XVI; septicæmia and pyæmia in Chapter X.

### **Treatment of bacterial inflammation**

The general principles of treatment may be summarized as follows:

#### **1. Local treatment**

(1) If the surface of the body is involved apply a sterile dressing to prevent further infection.

(2) Rest the part, if necessary using splints or other mechanical appliances.

(3) Apply heat. This promotes hyperæmia and absorption of inflammatory exudate; it also helps to relieve pain.

(4) Elevate the part. This improves the circulation by facilitating the return of venous blood to the heart, and helps to promote the absorption of inflammatory exudates.

(5) Evacuate localized collections of pus. This diminishes toxic absorption and greatly enhances the chances of resolution. *No general treatment such as the administration of an antibiotic is an effective substitute for surgery when pus is present*, though it may often advantageously be combined with incision.

(6) Remove any contributing cause such as a foreign body. This is mentioned after other methods of treatment because some foreign bodies cause no harm and, if deeply situated, are often best left alone unless they appear to be preventing resolution. Superficial foreign bodies which can be found easily should however be removed without delay.

(7) In certain cases of chronic inflammation apply counter-irritants such as iodine to the overlying skin, or treat the part with infra-red radiation or short-wave diathermy. This promotes hyperaemia and alleviates pain.

## 2. General treatment

(1) Put the patient to bed if there are severe general symptoms or if local rest and elevation cannot otherwise be achieved.

(2) Regulate the diet and the excretory functions.

(3) Treat any general cause of diminished resistance such as *diabetes mellitus*, loss of blood and so on.

(4) Institute 'specific therapy' in appropriate cases. This includes immunological procedures, chemotherapy and administration of antibiotics. Chemotherapy and antibiotics are dealt with in Chapter XI, but it will be convenient to give here a brief account of the immunological methods used in the treatment and prevention of infection.

As mentioned earlier, immunity may be innate or acquired. Acquired immunity may be *active* or *passive*.

Active immunity to many bacteria and bacterial toxins may be produced by previous infection, or by deliberate injection of the organisms or toxins in question. Injection of virulent living organisms or potent toxins, even in small amounts, may be dangerous; in practice, therefore, it is usual to employ instead either killed organisms (*vaccines*), organisms which have been treated in some way to reduce their virulence, or bacterial toxins treated in such a way as to render them no longer toxic though still capable of producing a state of immunity (*toxoids*). Active immunity takes some days to develop, and this type of immunization is therefore used for *prophylaxis* rather than for the treatment of established infection. Common



examples are the use of 'T.A.B.' vaccine for the prevention of typhoid fever, and tetanus toxoid for the prevention of tetanus.

Passive immunity is produced by injecting large amounts of serum from an animal (usually a horse) which has previously been actively immunized to the organism or toxin in question. Passive immunity develops at once, but is usually of short duration. This type of immunization is used in the *treatment* of certain bacterial diseases; anti-tetanic serum, for example, is used in the treatment of tetanus, and anti-diphtheritic serum in the treatment of diphtheria.

The injection of serum may cause dangerous allergic reactions in susceptible patients and in these who have received serum from the same species of animal on a previous occasion. The precautions taken to minimize the risk of this happening are described in the next chapter (p. 59).

## *Chapter V*

# INJURIES DUE TO MECHANICAL VIOLENCE, EXCLUDING FRACTURES AND JOINT INJURIES

Injuries due to mechanical violence may be subdivided according to whether there is or is not an external wound. Strictly speaking the term wound may be applied to all mechanically produced injuries, but in clinical practice it is usually restricted to cases in which there is a definite breach of continuity of the surface of the body (either skin or mucous membrane).

When the injury is severe *traumatic shock* and *haemorrhage* are likely complications. It will, however, be convenient to postpone consideration of the clinical features and treatment of these conditions to a later chapter (Chapter VIII).

### **Injuries without External Wound**

Injuries without any external wound may be classified as follows:

1. Contusions and haematomas.
2. Ruptured muscles and tendons.
3. Ruptured viscera.
4. Simple fractures.
5. Sprains, subluxations and dislocations.

We shall deal here with the first three groups in this classification and postpone consideration of fractures and joint injuries until the following chapter.

### **Contusions and haematomas**

A *contusion* is an injury due to mechanical violence in which there is damage to small blood vessels with consequent leakage of blood into the tissues. This leakage, or extravasation as it is sometimes termed, occurs usually into the subcutaneous tissues; sometimes, however, injury results in contusion of deeper structures, such as arteries, nerves and viscera.

Usually when contusion occurs the tissues are simply infiltrated with blood, but sometimes a circumscribed collection of blood is formed known as a *haematoma*.

*Diagnosis*

The diagnosis of a contusion of the subcutaneous tissues is obvious. The main symptoms and signs are pain, bruising (that is, discoloration due to chemical changes occurring in the extravasated blood) and swelling. If a haematoma forms it will usually be possible to demonstrate that the swelling is *fluctuant*.

Contusion of the deeper structures may be much more difficult to diagnose, but if severe it is usually associated with impairment of function of the structures involved. Thus, for example, contusion of the ulnar nerve may result in anaesthesia in the distribution of the nerve and paralysis of the muscles supplied by it.

*Sequelae*

The extravasated blood may be absorbed, but in some cases organization occurs resulting in a fibrous scar. Occasionally a haematoma becomes infected by pyogenic organisms circulating in the blood stream and an abscess is formed.

*Treatment*

The treatment of a superficial contusion or haematoma is simple. If the case is seen early extravasation may be limited by applying a firm bandage. Rest, both local and general, may be necessary. Heat should not be applied in the early stages as it causes vascular dilatation and may thus result in further extravasation. Later, however, both heat and massage may expedite absorption.

A localized haematoma is sometimes painful, especially if situated beneath the deep fascia, and in such cases aspiration of the blood through a needle, or even incision, may be required. If this is done, however, strict aseptic precautions must be taken.

**Haematomas of the scalp***Surgical anatomy*

The scalp consists of the following layers:

1. Skin.
2. Subcutaneous tissues, consisting of fat and strong septa of fibrous tissue.
3. *Galea aponeurotica*, a strong aponeurotic layer joining the *occipitalis* and *frontalis* muscles.
4. Layer of loose areolar tissue.

5. Pericranium; that is, the periosteum covering the vault of the skull. This is loosely attached to the bones but is firmly bound down at the suture lines.

The scalp is very freely supplied with blood vessels which ramify mainly in the subcutaneous tissue. The veins communicate via the emissary veins with the cerebral venous sinuses and as a result infection may spread inwards from the scalp and cause septic thrombosis and other serious intracranial suppuration.

The lymphatic drainage of the scalp is to the pre-auricular, post-auricular and occipital nodes, and thence to the deep cervical nodes.

#### *Types of scalp haematoma*

A blow which fails to break the skin may cause a subcutaneous, a sub-galeal or a sub-pericranial haematoma. A *subcutaneous haematoma* is rarely large because swelling and extravasation are limited by the fibrous septa which transverse the subcutaneous layer of the scalp. A *sub-galeal* (sometimes called a sub-aponeurotic) *haematoma* forms an extensive fluctuant swelling on the top of the head, extending from the occiput almost to the eyebrows, and laterally to the temporal ridge on each side. A *sub-pericranial haematoma* forms a localized swelling overlying one of the bones of the vault (usually the parietal), and limited by the firm attachment of the pericranium to the suture lines. Softening commonly occurs in the centre and the swelling then presents a firm raised periphery with a soft and depressed centre; it may be mistaken for a depressed fracture of the skull (Chapter VI).

Sub-pericranial haematoma sometimes occurs as a birth injury and is then known as a *cephalhaematoma*.

#### *Treatment*

No special treatment is required for a haematoma of the scalp unless, as occasionally happens, suppuration occurs due to secondary blood borne infection. In this event the pus must be evacuated and appropriate specific therapy instituted.

#### **Ruptured muscles and tendons**

Muscles or tendons are occasionally ruptured as the result of sudden muscular contraction; for example, the long head of the *biceps brachii* muscle, the *quadriceps femoris* muscle and the *Tendo Achillis*. In many cases repair by operation is necessary.

**Ruptured viscera**

It is not uncommon for viscera, especially abdominal viscera, to be severely torn as a result of mechanical violence, even in the absence of an external wound.

If solid abdominal viscera such as the liver, spleen and kidney are injured operative treatment may or may not be necessary. If operation is required its main purpose will be to arrest haemorrhage; this is achieved by suturing the tear or, in some cases, by removing the damaged viscus.

If hollow abdominal viscera such as the bladder, stomach and bowel are ruptured operation is necessary to mop up the urine, gastric contents or bowel contents which have escaped and prevent further leakage. If this is not done infection is sure to occur and is likely to prove fatal.

**Abrasions and Wounds**

Injuries due to mechanical violence in which there is breach of continuity of the skin or a mucous membrane may be classified in various ways:

1. *According to the causal agent* (gun-shot wounds, surgical wounds, wounds due to stings of insects and so on).
2. *According to the external appearance of the injured part* (abrasions, incised wounds, lacerated or contused wounds, punctured wounds).
3. *According to the deep structures involved* (wounds of arteries or nerves, abdominal wounds, compound fractures and so on).
4. *According to the presence or absence of bacterial infection.*

The subdivision of wounds into infected and non-infected is most important. It must be realized that these terms are only relative, since bacterial contamination occurs in some degree as a complication of practically all wounds. In the case of surgical and other 'clean' wounds, however, contamination is slight, the bacteria are rapidly destroyed and there may be no clinical evidence of infection.

Wound infection may occur at the time of injury, or later due to a faulty dressing technique. To minimize the risk of contamination dressing of wounds should never be undertaken within 2 hours of sweeping a ward (that is, before the dust has had time to settle), and the person doing the dressing should wear a mask and adhere to the aseptic technique described in Chapter II.

Where there is frank infection of a wound it is important to establish the identity of the predominating organisms. This may be apparent from clinical examination alone, but bacteriological confirmation by microscopic examination and culture of the exudate from the wound is always advisable and usually essential.

**Local treatment of abrasions and wounds, excluding compound fractures and joint injuries**

It will be convenient to consider separately six types of case:

*1. Abrasions*

Here the superficial layers of the skin alone are involved. The area is gently cleansed with cetrimide solution or some other non-irritant antiseptic such as proflavine or Dettol, and covered with a non-adherent dressing. Tulle gras, made by impregnating gauze with petroleum jelly, is often used for this purpose, but many surgeons prefer to use one of the non-greasy dressings which are now on the market.

*2. Small superficial and clean recent wounds*

If there is no evidence of damage to important structures, and the wound is small, superficial and apparently free from gross contamination, it is sutured forthwith.

As a general rule a prophylactic injection of anti-tetanic serum (1,500 units) should be given but this may be omitted in patients with a personal or family history of asthma, hay fever, infantile eczema or food allergy, provided that they have previously been actively immunized with tetanus toxoid (Chapter IV). If serum *has* to be given to a patient who gives an allergic history or who has previously been injected with horse serum, a trial subcutaneous injection of 0.2 ml. serum should be given and the patient should be kept under observation for half an hour. If there is no untoward reaction a second trial injection is given subcutaneously, and if once again there is no reaction the full dose of serum may be given intramuscularly. If, however, signs of anaphylaxis such as dyspnoea, cyanosis or increased pulse-rate occur the patient should be given a quick-acting anti-histaminic drug (such as mepyramine malleate 0.1g.) and amounts of not more than 0.2 ml. serum should be given subcutaneously every half hour. If these precautions are observed serious anaphylactic shock is unlikely to occur, but if it does the patient should be given 0.5 to 1.0 ml. of adrenaline (1 : 1,000 solution) by intramuscular injection. This solution should always be at hand when injections of serum are being given.

*3. Large, deep or contaminated recent wounds*

When shock has been treated (Chapter VIII) an anaesthetic is administered and the area round the wound is cleansed with soap and water or cetrimide solution, shaved if necessary and painted with antiseptic solution. The wound is then explored and the extent of damage to deep structures such

as large vessels, nerves and tendons is determined. Easily accessible foreign bodies are removed and all contaminated tissue is excised, including a narrow rim of skin except in certain special areas such as the face, palms and soles. Haemorrhage is arrested by clipping and ligating bleeding vessels; severed nerves or tendons are sutured if contamination is minimal, otherwise they are left for the time being and repaired at a later operation; a suitable antibiotic preparation, e.g. one of the proprietary mixtures of neomycin, polymyxin and bacitracin (Chapter XI), may be insufflated if considered desirable, and the wound is closed by sutures. A wounded limb is immobilized in plaster of Paris or by means of a splint. As a prophylactic measure the patient is given a single intra-muscular injection of anti-tetanic serum (at least 3,000 units) subject to the restriction and with the precautions discussed above, and a course of treatment with penicillin or some other antibiotic.

The process of cleansing a wound and removing contaminated tissue described above is known as *debridement*.

#### 4. *Sutured wounds which show subsequent evidence of infection*

The first thing to do is to relieve the tension in the wound by removing some or all of the sutures. A swab is taken for bacteriological examination so that appropriate antibiotic treatment can be started.

Any obvious foreign body which was left behind when the wound was sutured is removed, and pockets of pus are opened up. Apart from this no extensive operative treatment is indicated, except in two special cases; namely, sepsis of such severity as to threaten the life of the patient and gas gangrene.

Sepsis threatening life occurs most frequently when there has been a penetrating wound of a large joint with consequent suppurative arthritis. In such cases amputation is sometimes the only safe measure.

The treatment of gas gangrene will be described later (Chapter XII).

#### 5. *Contaminated wounds, first seen a considerable time after injury*

Excision of a wound (other than a trivial one) is almost always indicated if the patient is seen within 8 hours of injury, but after this period the chances of success greatly diminish. After 24 hours excision is almost certainly useless as by this time bacteria which were originally confined to the wound have extensively invaded the surrounding tissues. The wound is therefore simply opened up, any obvious foreign body is removed and the part is splinted. Sometimes it is useful to apply a complete plaster-of-Paris cast, discharge from the wound being allowed to soak into

the dressings. Occasionally immediate amputation is required for sepsis threatening life.

6. *Wounds in which all the main vessels and nerves to a limb are completely divided*

In these cases immediate amputation is indicated since there is no prospect of saving the limb.

### **Wounds of the scalp**

Wounds of the scalp are common. They may be produced in various ways and even when caused by a blunt instrument often appear to be clean cut. A special and very unpleasant type of injury is *avulsion of the scalp*; this is usually caused by the hair becoming entangled in machinery.

Scalp wounds gape widely if they extend deep to the galea, and even when superficial they bleed freely on account of the great vascularity of the scalp and because severed vessels are prevented from retracting by their attachment to the fibrous septa of the subcutaneous tissue.

The scalp is relatively resistant to infection, probably owing to its extensive blood supply.

Superficial wounds rarely suppurate, and can safely be sutured even when there has been a considerable contamination. Excision of the skin edges is seldom required.

In dealing with deep wounds caution is necessary. A careful examination must first be made to determine whether there is an underlying fracture of the skull, as this may require special treatment (Chapter VI). If there is no fracture and, in addition, the injury is comparatively recent and the degree of contamination slight, the wound may be sutured in two layers. The deeper layer of stitches unites the severed galea and the superficial one the skin and subcutaneous tissue. Haemostasis is usually readily achieved in this way, but if a large artery has been divided it may be necessary to ligate the two ends. If treatment has been delayed or the wound is heavily contaminated suturing should be postponed to avoid the risk of suppuration developing beneath the galea.

After avulsion of the scalp complete replacement is sometimes successful. It is preferable, however, to cut thick split-skin grafts (Chapter IX) from the detached portion and apply these to the raw area. Failing this, extensive skin grafting will be necessary later to obtain an epithelial covering, and the patient may require a wig to conceal the bald area.



**Wounds of the face and lips**

Wounds of the face and lips bleed freely, but usually heal rapidly. Accurate repair with early removal of sutures (usually after about 3 days) is essential if a good cosmetic result is to be obtained.

A non-absorbable suture material such as fine silk is used for the skin and catgut for the mucous membrane.

## *Chapter VI*

# FRACTURES

A fracture may be defined as a sudden breach in the continuity of a bone.

Fractures are caused by mechanical violence. This may take the form of direct external violence, indirect external violence or muscular action.

Fractures due to direct external violence occur at the site of impact; those due to indirect violence occur some distance away. A blow on the head with a hammer, for example, may cause a fracture of the vault of the skull by direct violence; a fall on the outstretched hand may result in a fracture of the radius, humerus or clavicle by indirect violence. Fractures due to muscular action occur commonly in the patella as a result of sudden contraction of the quadriceps muscle in attempting to avert a fall.

Considerable force is usually necessary to produce a fracture but if there is pre-existing disease of bone a very slight degree of violence may suffice. In such cases the term *pathological fracture* is used.

### **Classification of fractures**

Fractures are subdivided into *closed* (or *simple*) and *open* (or *compound*) fractures. An open fracture is one which communicates with the surface of the body through a wound in the overlying skin or mucous membrane. Such a wound may be produced by the external force which causes the fracture, in which case the fracture is said to be compound from without inwards; alternatively the wound may be caused by one of the bone ends, when the fracture is said to be compound from within outwards.

In both simple and compound fractures there may be injury to other structures such as joints, nerves, blood vessels or viscera, and the fracture is then described as *complicated*.

Fractures may also be classified according to the configuration of the bone ends at the fracture site. If the continuity of the bone is only partially interrupted the fracture is termed *incomplete*, otherwise it is *complete*. Complete fractures are described as *transverse*, *oblique*, *longitudinal* or *T shaped* according to the direction of the fracture line in relation to the long axis of the bone; as *impacted* or *non-impacted* according to whether or not one fragment is driven into the other; and as *non-comminuted* or *comminuted* according to whether the bone is broken into two, or more than two, pieces.

Yet another subdivision may be made according to the type of deformity. This depends on the nature and direction of the violence responsible for the fracture, the direction of the plane of the fracture, the pull exerted on the bone fragments by muscles attached to them and in some cases also on gravitational forces. It may include angulation, lateral displacement, shortening due to impaction or overlap of the bone ends, and rotation.

In young people injury commonly occurs in the region of the epiphyseal line near one or other end of a long bone. Such an injury is commonly spoken of as a juxta-epiphyseal fracture.

### **The repair of fractures**

Three stages may be distinguished in the repair of a fracture :

*Stage 1. Repair by granulation tissue.* A haematoma forms between the bone ends and gradually becomes organized. This stage is usually complete in a few weeks with large bones like the femur, and rather more quickly in the case of smaller bones such as metacarpals or phalanges.

*Stage 2. Repair by primary callus.* Minute bony trabeculae grow from the bone ends into the granulation tissue. Calcification, chondrification and ossification occur in a somewhat patchy manner so that the bone ends come to be welded together by a tumour-like mass of bone and cartilage. This stage, in the case of a large bone, usually lasts about two months.

*Stage 3. Final consolidation or union of the fracture.* The primary callus is gradually replaced by bone having a proper lamellar structure. The original contour of the bone is restored by a combination of absorption and new bone formation.

The rate of repair of a fracture may be influenced by a number of factors ; namely :

1. *The age of the patient.* Repair is more rapid in young patients than in old. The shaft of the femur, for instance, may be completely repaired in a month in a new-born baby, but may take six months or more in an adult.

2. *The general health and nutrition of the patient.* General diseases and debility delay repair. Dietetic factors may be important ; lack of vitamin D, for example, results in imperfect calcification.

3. *The type of fracture.* Repair occurs more rapidly when the fracture surface covers a large area. Thus a long oblique fracture often unites more quickly than a transverse one.

4. *The gap between the bone ends.* Good repair is possible only if there is a continuous haematoma between the bone ends. A large gap takes a long time to bridge and the interposition of muscle or other soft tissues may delay healing indefinitely.

5. *The particular bone involved.* This is mainly a matter of blood supply, healing being slow in sites such as the lower third of the tibia, where the blood supply is poor, and rapid when the blood supply is abundant.

6. *The presence of infection.* Infection occurs in many compound fractures and delays union.

7. *The degree of immobilization.* Inadequate immobilization delays union and may prevent it altogether.

8. *Unexplained individual variation.*

### Symptoms and signs

The patient gives a history of injury and sometimes says he has heard or felt the bone crack. He complains of pain and usually also of loss of function, though the latter symptom may not be noticed at first with impacted fractures.

On examination the following signs may be present:

1. Tenderness of the bone on pressure.
2. Swelling and bruising.
3. Blebs in the skin overlying the fractured bone.

These often appear some days after the fractures in cases where reduction has not been carried out.

4. Deformity. This is one of the most important signs of fracture. It is elicited by inspection, palpation and measurement; in fractures of a limb the corresponding uninjured limb is used as the standard of comparison.

The deformity in the case of a long bone may be angular, lateral longitudinal (shown by shortening of the bone) or rotatory; and in the case of a flat bone there may be depression or elevation.

5. Abnormal mobility.

6. Crepitus.

Abnormal mobility and crepitus are absent in the case of impacted fractures. In seeking to elicit these signs great gentleness must be exercised.

7. Signs of loss of function. If ribs are fractured, for example, the patient may be unable to take a deep breath; if the femur is fractured he will be unable to bear weight on the affected limb.

In all cases of suspected fracture radiographs are taken to confirm or refute the diagnosis, and to give the precise information about the position of the bone ends which is necessary for successful reduction.

Films should always be taken in two planes at right angles (usually anteroposterior and lateral) and sometimes oblique views are required as well.

During the period of treatment further films are taken from time to time to assist in determining progress.

**Treatment of simple fractures**

The treatment of an uncomplicated simple fracture falls under three main headings: *reduction, retention and restoration of function.*

*Reduction*

If displacement is negligible reduction is not required; otherwise an attempt is made to obtain perfect anatomical reposition of the fragments. In practice this is not always possible, but to achieve a good result in fractures of the long bones it is essential to obtain correct length and alignment, and freedom from rotational deformity.

Reduction is effected by manipulation, skeletal traction or open operation.

*Reduction by manipulation.* The first essential is muscular relaxation. This is usually best obtained by giving a general anaesthetic, but sometimes a local anaesthetic is injected into the fracture haematoma.

If the fracture is impacted it must be forcibly disimpacted. Overlap, if present, is corrected by traction in the long axis of the bone against countertraction applied by an assistant. Rotational deformity is then corrected and the bone ends are manipulated into apposition. Finally, angulation is corrected, and this is facilitated if, as often happens, the periosteum remains intact over one aspect of the fracture and can thus act as a hinge during the process of reduction.

*Skeletal traction.* As described below, skeletal traction is used for retention of certain fractures after reduction by manipulation. It is also used sometimes as a method of reduction.

A stainless steel pin is driven through the bone distal to the fracture and traction is applied to the pin by a system of weights and pulleys. Countertraction may be provided by the patient's own weight or by means of a second pin driven through the bone proximal to the fracture.

*Reduction by open operation* is used when simpler methods are ineffective. The fracture is exposed and the bones are levered into correct position.

*Retention*

After reduction the bone ends are retained in position by external splinting, traction and countertraction, multiple fixation pins or internal fixation.

*External splinting* is usually in the form of plaster of Paris. Occasionally, in trivial cases, a simple bandage suffices. Plaster of Paris is applied in the form of plaster-impregnated bandages which are available from a number of manufacturers. They are stored in dry tins and are soaked in water for a few seconds before use.

Slabs several layers thick are made on a table, then applied to the limb and bound in place with encircling turns of bandage. A plaster cast, if properly applied, provides excellent immobilization.

*Traction and countertraction* is useful where, owing either to the pull of strong muscles or the obliquity of the fracture surfaces, external splinting is insufficient to prevent re-displacement. Two methods are available: skin traction and skeletal traction.

Skin traction is effected by strips of foam rubber applied to the skin; skeletal traction by one or more stainless steel pins driven through the bone.

When using skin traction, or skeletal traction with a single pin driven distal to the fracture, the foot of the bed is raised so that countertraction is provided by the weight of the patient's body.

*Multiple transfixing pins.* Multiple transfixion pins are becoming obsolete, though they are still occasionally used for the treatment of mandibular fractures.

*Internal fixation* may be effected by means of a stainless steel pin, nail or screw (Figures 19–21), a bone graft, a metal plate or a suture passed through or round the bone.

Internal fixation is commonly used in conjunction with operative reduction of a fracture; it is usually supplemented by some form of external fixation.

Whatever method of retention is used it is usually necessary, with fractures of the long bones, to immobilize the joint above and the joint below the fracture.

Immobilization is continued until there is clinical and radiological evidence of union.

*Clinical evidence of union.* Try by gentle manipulation to bend the bone at the site of fracture. If this causes no pain and the bone is unyielding, repeat the procedure using a *little* more force. If the result is the same there is clinical evidence of union.

*Radiological evidence of union.* The radiographs must be of good quality. Films taken through plaster, though of value for checking the reduction of a fracture, are useless for determining whether union has taken place.

The essential sign of union is continuity of bone across the line of fracture. It is *not* necessary for all trace of this line to have disappeared; sometimes, especially in mandibular fractures, it can still be seen years after the fracture has united.

### *Restoration of function*

All the joints of a fractured limb, except those deliberately immobilized, must be exercised regularly, beginning within 24 hours of injury. This



Fig. 19

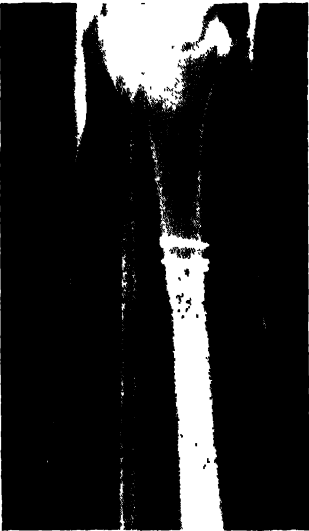


Fig. 21

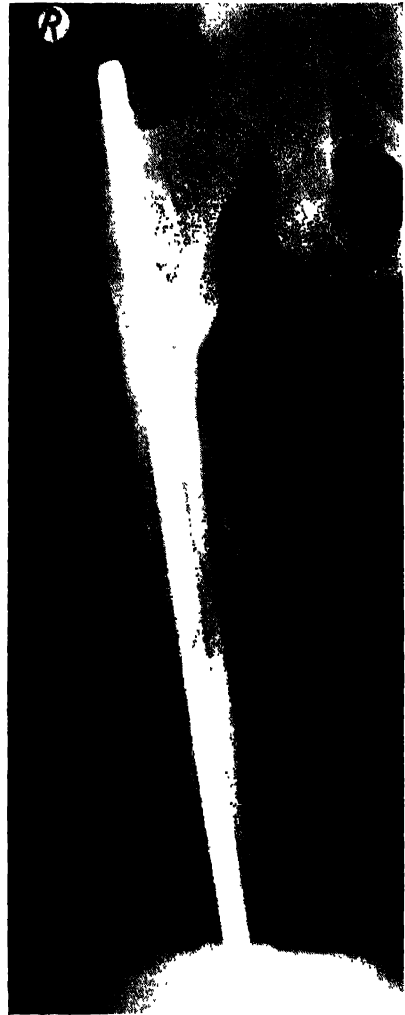


Fig. 20

FIG. 19. Radiograph showing a fracture of the neck of the femur treated with a Smith-Petersen pin.

FIG. 20. Radiograph showing internal fixation of a fracture of the shaft of the femur with a Küntscher nail (Prof. J.I.P. James' case).

FIG. 21. Radiograph showing fixation of a fracture of the tibia with two metal screws (Prof. J.I.P. James' case).

limits wasting of muscles and helps to avoid subsequent stiffness in the joints.

As soon as the fracture has united active movements must be started in the joints which have been immobilized, in order to restore normal mobility. Passive movements may do more harm than good and should be avoided.

### **Treatment of compound fractures**

As already stated, a fracture may be compound from without inwards or from within outwards. Severe infection is more likely to occur in fractures compound from without inwards but the principles of treatment are the same in both types; namely:

1. Treatment of the wound, to prevent or combat infection and to promote healing.
2. Treatment of the fracture.

The procedure depends on the interval between the injury and the time at which treatment can be undertaken; and also, to some extent, on the site of the wound.

#### *Relatively recent cases*

Normally the patient arrives in hospital within a few hours of injury. In this event the wound is excised (Chapter V) and the skin is sutured; the open fracture is thus converted into a closed one, and is henceforth treated as such. It is probably beneficial to insufflate a topical antibiotic preparation, e.g. one of the proprietary mixtures of neomycin, polymyxin and bacitracin, into the wound before closing the skin. Sulphonamide powder should however not be used.

In excising the wound an exception is made of the bone ends; these may be cleaned and any loose bits of bone may be removed, but deliberate excision of the bone ends is not practised.

Sometimes during the operation advantage may be taken of the exposure of the bone ends to reduce the fracture. It is not wise, however, to use any form of internal fixation in compound fractures owing to the risk of subsequent sepsis.

When the wound is situated on the face excision of the skin edges is omitted.

In the special case of fractures which are compound only into the mouth debridement of the wound is not undertaken. If the mucosa is severely torn, however, it is sutured and every effort is made to cover any exposed bone. Frequent irrigations are prescribed to help control infection.



*Cases not seen early*

In military surgery, and very occasionally in civilian practice also, the patient may not be seen until 12 or more hours after injury, and the outlook is much less favourable. Wound excision is useless and sometimes harmful, and the main item in the treatment, after opening up the wound to relieve tension and provide an outlet for pus, is fixation. Reduction of the fracture may be attempted, but if difficulty is encountered the attempt should be abandoned, the wound lightly packed with vaseline gauze and the limb immobilized. Antibiotics are given systemically. It is reasonable to begin with penicillin, or a combination of penicillin and ampicillin, but swabs should be taken daily from the wound for bacteriological culture and the antibiotics should be changed if necessary in accordance with the sensitivities of the predominant organism. It is often good treatment to enclose the limb in a complete plaster and—unless the patient's general condition deteriorates, the plaster becomes softened by soakage of blood and pus or the odour becomes unbearable—leave it undisturbed for several weeks. This method was first described by Winnet Orr; it was used by Trueta during the Spanish civil war and by many surgeons during the Second World War.

When treatment is delayed the risk of infection is correspondingly increased. At the very least union of the fracture is likely to be retarded and frequently osteomyelitis develops. Occasionally infection is so severe that early amputation is necessary.

**Complications of fractures**

Some of the complications of fractures have already been mentioned but others remain to be described. The following scheme summarizes the main complications which may occur.

**1. General complications**

Shock.

Haemorrhage.

Delirium Tremens.

Bronchopneumonia.

Fat Embolism.

General Infection—pyaemia or septicaemia.

**2. Local complications**

Bones. Infection.

Avascular necrosis.

Delayed union and non-union.

Malunion.

- Joints.** Injury at the time of the fracture.  
Stiffness due to immobilization.  
Osteoarthritis.
- Muscles.** Disuse atrophy.  
Myositis ossificans.

Injury to blood vessels, nerves or viscera.

*Shock* and *haemorrhage* are discussed in Chapter VIII; *pyaemia* and *septicaemia* in Chapter X. *Delirium tremens* occurs in alcoholics. *Broncho-pneumonia* is common after severe injuries in elderly patients.

*Fat embolism* is a condition in which particles of fat from the bone marrow enter the circulation and cause blockage of small arteries in the lungs, brain and other organs. Minor degrees of fat embolism without symptoms are common. Severe cases are rare and usually fatal. Symptoms appear a few hours to a few days after injury; they include fever, rapid pulse and respiration, cough, cyanosis, excitability and convulsions or coma. There is no specific treatment, but administration of oxygen may help and sedatives should be given unless the patient is comatose.

*Bone infection* (osteomyelitis) will be dealt with later (Chapter XIX).

*Avascular necrosis*—that is, death of tissue as a result of deficient blood supply—often occurs in small fragments of bone in comminuted fractures. It may also occur in larger portions of bone whose blood supply is cut off as the result of a fracture; a common example is avascular necrosis of the head of the femur following fracture of the neck of this bone.

The term *delayed union* implies that repair is proceeding along normal lines but is taking longer than usual. This may be due to: (a) *general causes*, such as debility and old age; (b) *local infection*; and (c) *improper treatment*, especially excessive traction, incomplete immobilization or immobilization for too short a period. It is particularly likely to occur in situations such as the lower end of the tibia where the blood supply of the bone is poor.

The condition is recognized by clinical and radiological examination. In the radiographs the bone ends are of normal density or show a certain amount of decalcification and there is no continuity of bone across the fracture line. Union may occur if immobilization is sufficiently prolonged without any other treatment, but much time can usually be saved by bone grafting. Grafts in the form of thin strips are cut from the patient's iliac crest. The fractured bone is exposed and the periosteum is raised proximal and distal to the fracture line without disturbing the site of the fracture. The iliac bone is then packed into the subperiosteal space, the wound is closed, and immobilization is continued until union occurs.

The term *non-union* implies that repair is not proceeding along normal lines *and that without special operative treatment the fracture will never unite.*

There are two possible causes of this condition : (a) absence of a continuous haematoma between the bone ends, due to gross loss of bone substance or the interposition of soft tissues; and (b) inadequate immobilization.

The great majority of cases are due to inadequate immobilization. Radiological examination shows that the bone ends are rounded and *sclerosed* (that is, abnormally dense), and if the fracture is exposed at operation it is seen that the space between the bone ends is occupied by dense fibrous tissue.

Treatment is by bone grafting as already described for the treatment of delayed union.

After operation immobilization is necessary until union occurs.

*Malunion* means union in a faulty position. If the degree of malunion is slight, treatment may not be necessary. If there is gross malunion the bone must be refractured, the deformity corrected, and immobilization in correct position maintained until satisfactory union occurs.

*Joint stiffness* is sometimes inevitable following immobilization of a fracture. Except in old people, however, it is usually only temporary, provided due precautions have been taken against it. These precautions have already been mentioned; namely, (a) active movement *ab initio* of all joints not requiring to be immobilized; (b) active movement of the joints which have been immobilized as soon as the splints are discarded; and (c) avoidance of forcible passive movements at all stages of treatment.

*Osteoarthritis* will be discussed later (Chapter XX).

*Disuse atrophy of muscles* can be minimized by taking the precaution described above for the prevention of joint stiffness.

*Myositis ossificans traumatica* is a condition in which ossification develops in soft tissues, particularly muscle, in relation to a fracture. It occurs commonly as a complication of fractures in the neighbourhood of the elbow joint.

*Injury to blood vessels and nerves* will be discussed later (Chapters XVII and XXI). *Injuries of viscera* are beyond the scope of this book.

## Fractures of the Mandible

The treatment of fractures of the jaws will be taught in detail during the course on clinical dental surgery. Our present purpose is to illustrate the general principles already enumerated by reference to the special case of mandibular fractures.

Fractures may occur in any part of the mandible and are frequently bilateral. They are usually the result of violence applied while the muscles—temporal, masseter and pterygoids, on each side—are relaxed. Under such conditions a blow on the point of the chin may lead to fractures of the necks of both condyles by indirect violence, and sometimes also to a fracture in the region of the symphysis by direct violence. A blow on the side of the jaw commonly causes a fracture by direct violence at the site of the impact and another by indirect violence on the opposite side, usually at the angle or the neck below the condyle. Fractures near the angle of the jaw or in the ascending ramus of the bone are quite common; fractures at the base of the coronoid process are relatively rare and occur only as a result of direct violence.

Pathological fractures of the jaw are uncommon, but sometimes occur as the result of excessive alveolar resorption, osteomyelitis, cysts or malignant tumours.

The *displacement* depends, as stated earlier, on the nature and direction of the violence, the direction of the plane of the fracture, the pull of muscles and gravity. An additional factor of importance is the presence or absence of opposing teeth.

The genio-hyoid muscles, attached to the body near the symphysis, pull downward and backward; the masseter, attached to the ascending ramus, and the temporal, attached to the coronoid process, pull upward; the medial pterygoid, attached in the region of the angle, pulls forward, upward and inward. Hence, if there is a fracture of the body in the molar region, the posterior fragment tends to be rotated upward and inward. Upward displacement may, however, be largely prevented if the fracture plane runs from above downward and forward, or if there are opposing teeth in the upper jaw and the short posterior fragment; inward displacement is minimal if the fracture plane runs forward and outward.

### *Clinical features*

There is a history of injury with pain, swelling, bruising and deformity. Deformity is often most apparent on inspection of the teeth. Movement of the jaw is limited and loss of function occurs in the form of difficulty in speaking and swallowing. There is sometimes a considerable degree of trismus.

Fractures involving the body of the mandible in edentulous patients are usually simple, but in dentulous patients they are nearly always compound into the mouth, and when this is the case there may be dribbling of blood-stained saliva.

Sometimes, as a result of gun-shot injury, fractures of the mandible occur which are compound externally; these, however, are rarely seen in civilian practice.

In all cases radiographs are taken using three standard projections: posterior-anterior, right and left lateral oblique. Sometimes additional special views are required; it may, for instance, be necessary to use intra-oral films to determine the exact relation of tooth roots to the fracture.

## **Principles of treatment**

### *First aid*

The first aid treatment may be summarized in a sentence: Maintain a free airway, control haemorrhage and treat shock.

*Maintenance of an airway* is of vital importance. If the patient is walking he should be told to keep his head forward. If he is carried on a stretcher he should, if possible, lie face down with his forehead supported in the bend of one arm; failing this his tongue should be held forward by means of a suture inserted through its tip.

*Control of haemorrhage* is discussed in Chapter VIII. Severe haemorrhage is a rare complication of mandibular fractures in civilian life, but is more common in war surgery.

*Treatment of shock.* The patient should be kept reasonably warm by being wrapped in a blanket but should not be overheated with hot water bottles. Morphia and related analgesics which exert a central respiratory depressant effect are dangerous in patients with any impairment of the airway and should be avoided so far as possible.

*Should the jaw be immobilized?* Many books on first aid recommend the use of a four-tailed bandage. This is bad advice. A four-tailed bandage can only do harm because it forces the chin backwards and thereby increases displacement or perhaps creates displacement where none existed. If a bandage is used it should be applied in such a way as to hold the mandible upward and forward.

### *Subsequent treatment*

If the fracture is stable and there is no displacement, no special treatment is needed beyond advising the patient to take a soft diet for two or three weeks. In other cases reduction and immobilization must be undertaken as with fractures elsewhere. A general anaesthetic is usually necessary though the preliminary taking of impressions, if required, may be done without an anaesthetic.

All soft tissue wounds should be debrided and closed in layers, care being taken to cover all exposed bones.

It is often advised that a tooth in the line of the fracture should be extracted but, if intra-oral films show that the tooth in question is intact, it may be worth trying to conserve it. Furthermore, it may sometimes pay to retain even a damaged tooth temporarily if its presence contributes to the stability of the fracture or makes fixation easier.

The method of immobilization depends on the type and site of the fracture, and on the extent of displacement of the fragments. The following procedures are available and may be used singly or in combination:

Patient with teeth	Edentulous patient
1. Dental wiring methods.	1. Patients own dentures or Gunning splints.
2. Cast metal cap splints.	2. Gunning splints + circumferential and peri-alveolar wires.
3. Extra-oral pin fixation.	3. Extra-oral pin fixation.
4. Direct transosseous wires.	4. Direct transosseous wires.

In many cases intermaxillary fixation is necessary by means of a bandage or by intermaxillary fixation of splints. Immobilization is usually maintained for 4–6 weeks depending on the age of the patient except in the case of fractures of the condylar process where 10–12 days will normally suffice.

It is extremely important to maintain oral hygiene. This entails routine tooth brushing, frequent cleansing mouthwashes and irrigations with bicarbonate of soda, and where necessary careful mopping of the gingival and buccal mucosa with cotton wool sticks.

Feeding of maxillo-facial cases presents problems which vary according to the type of fixation employed. All cases require a soft diet and those with intermaxillary fixation have to be maintained on an almost fluid diet. The task of providing a suitable balanced diet of sufficient calorific value is facilitated by using a food-mixing machine and by including concentrated foods such as Complan.

The common methods of feeding are as follows:

1. Cup or bowl feeding.
2. Spoon feeding.
3. Drinking tube feeding.
4. Feeding through a naso-gastric tube.

The rare case of mandibular fracture due to a gunshot wound, which is compound externally, is treated according to the same principles as a compound fracture elsewhere. If the patient is seen early the wound is

excised. There is often considerable loss of tissue in these cases and if the defect in the soft tissues cannot be closed the mucosa is sutured to the skin as a temporary measure. Later, when infection has subsided, a plastic operation is undertaken on the soft tissues, and the defect in the bone is made good by a bone graft.

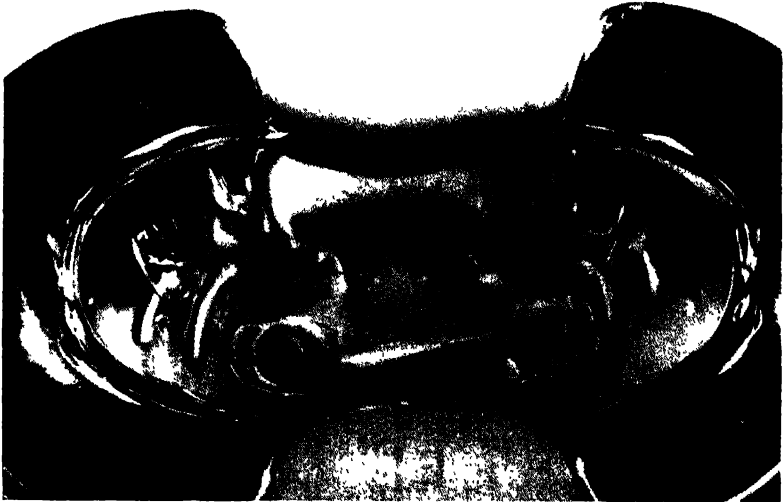


FIG. 22. Cast metal cap splints upper and lower jaw (Dr W.D. MacLennan's case).

### *Complications*

Serious general complications are not common with fractures of the mandible which are properly treated, but inhalation pneumonia develops occasionally in severe cases.

The important local complications are malunion, delayed union and non-union of the fracture, osteomyelitis of the mandible, and anaesthesia of one-half of the lower lip due to injury to the inferior alveolar nerve.

*Malunion* is due to immobilization in faulty position. If the deformity is severe *osteotomy* (that is, division of the bone) may be necessary, followed by immobilization in correct position until union occurs.

*Non-union* is rare except in the case of externally compound fractures with extensive loss of bone and severe infection. Should it occur a bone grafting operation is done when the infection has subsided. The graft is usually taken from the crest of the ilium; it may be applied in one piece, but many surgeons now prefer to cut it up into *chips* about the size of the little finger nail.

*Osteomyelitis* requires prolonged immobilization, drainage of abscesses and removal of dead teeth and bone sequestra.

### **Fractures of the Upper Jaw**

The simplest classification of fractures involving the upper jaw is that of Le Fort, which recognizes three categories:

Class 1. Fractures of the maxillary complex (Guerin's fracture).

Class 2. Fractures of the maxillary complex plus the nasal complex.

Class 3. Fractures of the maxillary complex plus the nasal complex plus the malar complex on one or both sides.

Most of these fractures are compound and they are often comminuted.

The symptoms and signs of upper jaw fractures are in general similar to those of fractures of the mandible; particular signs include excessive mobility, or impaction associated with an anterior open bite. The special features of fractures of the malar complex are discussed below. There may be an associated fracture of the base of the skull in the anterior fossa resulting in cerebrospinal rhinorrhea; this sign may not appear, however, until up to 48 hours after injury.

#### *Treatment*

Reduction and immobilization should be undertaken as soon as possible after injury to avoid malunion and also because the patient's general condition usually improves rapidly when this has been done. Fixation is usually provided by skeletal wires or by intra-oral splints connected to a plaster of Paris headcap (Fig. 24).

Depressed fractures involving the antrum are usually associated with fracture of the malar bone; they are discussed below.

### **Fractures of the Malar and Related Bones, including the Zygomatic Arch**

Fractures of the malar bone are often associated with fractures of the maxilla and other bones of the face. There may, for instance, be involvement of the malar and maxillary portions of the orbital margin, or of the malar bone and the maxillary antrum. These fractures are always due to direct violence and the bone is usually depressed and sometimes severely comminuted. The symptoms and signs vary according to the severity of the injury, but may include swelling (Fig. 23) (especially around the orbit), bruising, subconjunctival haemorrhage, visible and palpable bony deformity, diplopia associated with depression of the floor of the orbit, anaesthesia



in the distribution of the infra-orbital nerve, bleeding from the nose on the affected side, surgical emphysema and radio-opacity of the maxillary sinus on the affected side. In fractures of the zygomatic arch there is local pain and tenderness, visible depression at the site of the fracture and sometimes limitation of movement of the mandible due to pressure on the coronoid process.



FIG. 23



FIG. 24

FIG. 23. Fracture of bones of middle third of face showing extensive peri-orbital swelling (Dr W.D. MacLennan's case).

FIG. 24. Fracture of upper jaw immobilized by intra-oral splints connected to a plaster of Paris headcap (Dr W.D. MacLennan's case).

### *Treatment*

Reduction can usually be effected by the method of Gillies. An incision is made above the hair-line in the temporal fossa and a Kilner lever is passed down deep to the fascia covering the temporal muscle. Alternatively, a curved hook is inserted under the edge of the bone through a minute horizontal incision on the face just anterior to the masseter muscle. If, however, reduction cannot be achieved in this way or the fracture remains unstable a modified Caldwell-Luc operation (Chapter X) is performed, the bone is elevated from within the maxillary sinus and a pack is placed in the sinus and kept in place in the sinus for three or four weeks to act as a support. Alternatively, extra-oral pin fixation or direct trans-osseous wiring may be employed. In compound fractures associated with soft

tissue wounds of the face the fracture should always be reduced before the soft tissues are sutured.

### Fractures of the Skull

In many patients with fractures of the skull the brain is damaged at the time of injury or becomes subject to pressure due to the formation of a haematoma between the bone and the dura mater, or under the dura, or within the substance of the brain. In such cases the cerebral condition dominates the clinical picture and largely determines the treatment. The subject of brain injuries is beyond the scope of this book, but we shall give a brief classification of skull fractures and indicate the treatment so far as the fracture is concerned.

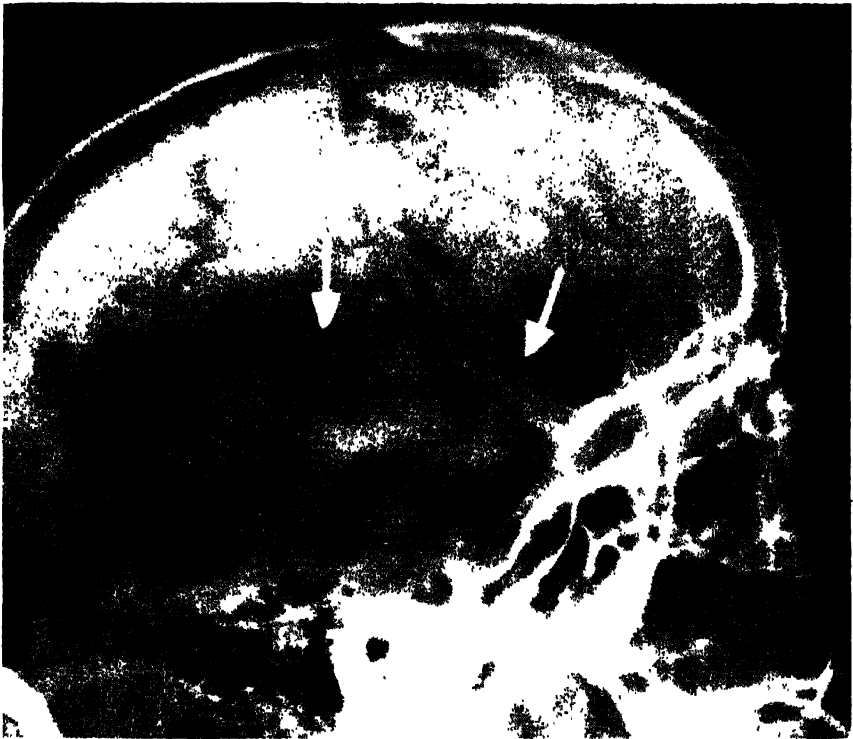


FIG. 25. Radiograph showing a linear fracture of the skull.

### Classification

Fractures of the skull may involve the vault or the base.

*Fractures of the vault* may be simple or compound, and in either case may be linear, star-shaped, comminuted or depressed. Except in children,

however, depressed fractures are nearly always compound. Radiographs of a linear and a depressed fracture are shown in Figures 25 and 26 respectively.

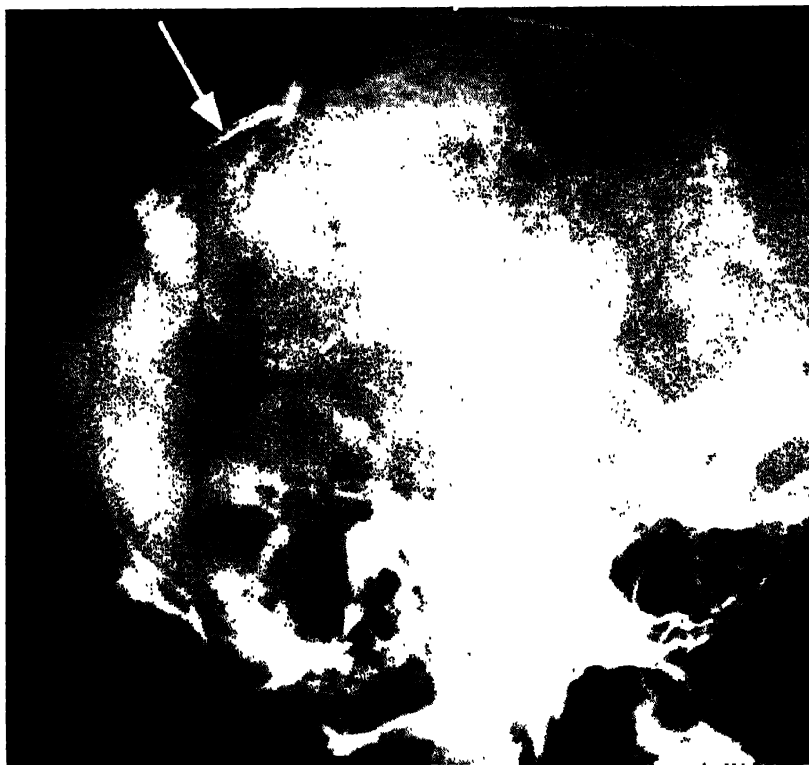


FIG. 26. Radiograph showing a depressed fracture of the skull.

*Fractures of the base* are subdivided on an anatomical basis into fractures of the anterior, middle and posterior fossae. The vast majority are compound into the nose, ear or pharynx, and, as a result, there is often escape of blood or cerebrospinal fluid—to the nose from the anterior fossa, to the ear from the middle fossa and to the pharynx from the posterior fossa. Alternatively, blood from an anterior fossa fracture may appear round the orbit and beneath the conjunctiva covering the eyeball, and from the posterior fossa may form a haematoma behind the mastoid process. In the former case the condition must be distinguished from a simple 'black eye'. This is not difficult because, in a fracture of the anterior fossa, the blood spreads forward from the back of the orbit so that there is no posterior border to the subconjunctival haemorrhage, and the area of

bruising is sharply limited to the orbital margin. In a black eye, on the other hand, if subconjunctival haemorrhage occurs it shows a sharp posterior margin and the bruising round the eye encroaches on the face and forehead.

In basal fractures there is often associated damage to cranial nerves. This is scarcely surprising in view of the large number of nerves which traverse foraminae in the base of the skull.

**Treatment** (excluding treatment of the brain injury)

Simple fractures of the vault do not require special treatment except in the rare case of a depressed fracture when elevation of the depressed fragment is usually necessary.

Compound fractures of the vault are treated according to the same principles as compound fractures elsewhere. In recent cases the wound is excised, foreign material is removed and depressed fragments of the bone are elevated. If the dura mater is opened infection may be carried in from the wound and cause septic meningitis. If, therefore, the dura is intact it should be left severely alone unless a collection of blood beneath it requires to be evacuated.

With fractures of the base meningitis may develop owing to infection from the nose or ear and steps must be taken to minimize this danger. If there is bleeding or discharge of cerebrospinal fluid into the nose the patient, as soon as he becomes conscious, is told not to blow his nose; later, if the discharge persists, operation may be required to seal the defect in the skull. Bleeding or discharge of cerebrospinal fluid to the ear usually ceases spontaneously and the only local treatment needed is to cover the ear with a sterile pad. In every case the patient is given penicillin or one of the other antibiotics as a prophylactic measure.

## *Chapter VII*

# INJURIES OF JOINTS

### **The Structure of a Synovial Joint**

Most of the joints in the body, including all those which possess a wide range of movement, are of the type known as *synovial* and are fundamentally similar in structure.

In most cases two, but sometimes three or more, bones enter into the articulation. The bones are united by a *joint capsule*, which is a sleeve of connective tissue lined by *synovial membrane*. The articular ends of the bones are covered with smooth hyaline cartilage.

The fibrous part of the capsule is reinforced, in regions subjected to special stress, by bands of tough connective tissue known as *ligaments*. In some joints there are, in addition, intra-articular ligaments which stretch across the joint cavity. The synovial membrane is composed of vascular connective tissue lined by endothelium. It secretes an oily fluid which lubricates the joint.

Some joints are provided with discs of fibro-cartilage which separate the articular ends of the bones. The knee and temporo-mandibular joints are in this category.

The stability of a synovial joint depends partly on the shape of the bone ends, partly on the ligaments and partly on the muscles which move the joint.

### **The temporo-mandibular joint**

The bones taking part in the temporo-mandibular joint are the condyle of the mandible, and the glenoid fossa and articular eminence of the temporal bone. There is a complete fibrous capsule, thickened on the outer aspect of the joint to form the *temporo-mandibular ligament*, and there are two accessory ligaments distinct from the capsule. These are the *spheno-mandibular ligament* attached to the spine of the sphenoid and the lingula, and the *stylo-mandibular ligament* which is a thickened band of cervical fascia attached to the styloid process of the temporal bone and the angle of the mandible.

A disc of fibro-cartilage—the *articular disc*—separates the joint into upper and lower compartments, each with its own synovial membrane

(Fig. 27). The upper surface of the disc is concavo-convex; the lower concave. The disc is thin where it articulates with the postero-inferior surface of the articular eminence and thickest a little behind this point where it occupies the deepest part of the glenoid fossa. It is attached all round its circumference to the capsule and anteriorly some fibres of the lateral pterygoid muscle are inserted into it.



FIG. 27. Microradiographic section of the mandibular joint.

- |                                  |   |
|----------------------------------|---|
| A. Mandibular condyle.           | E. Fatty and vascular tissue.           |
| B. Eminentia articularis.        | F. Upper joint cavity.                  |
| C. Fibro-cartilaginous covering. | G. Lower joint cavity.                  |
| D. Inter-articular disc.         | H. Fibres of external pterygoid muscle. |
|                                  | I. External auditory meatus.            |

(Reproduced from the 'British Journal of Radiology' by kind permission of the editor and Dr William Campbell.)

The joint is supplied by the auriculo-temporal and masseteric branches of the mandibular division of the trigeminal nerve.

The movements which occur at the joint are depression, elevation, protrusion, retraction and lateral movement of the mandible. *Depression* is produced by the digastric, geniohyoid and lateral pterygoid muscles of both sides acting together, and by the mylohyoid muscle. Movement from the position of occlusion to the rest position is brought about by rotation of the condyle in the lower compartment of the joint about a horizontal

axis. Beyond the rest position the movement is more complex. In the upper compartment the disc glides forward and downward on to the articular eminence, and at the same time the condyle continues to rotate in the lower compartment. As the position of maximum opening is approached the gliding movement of the disc becomes increasingly important and the rotation of the condyle diminishes. *Elevation* of the jaw is produced by the masseter, temporal and medial pterygoid muscles of both sides acting together. The movements which take place at the joint are the reverse of those occurring during depression. *Protrusion* is effected by the medial and lateral pterygoids of both sides acting together, and *retraction* by the posterior fibres of the two temporal muscles. These movements take place between the articular disc and the temporal bone. *Lateral movement* is effected by the medial pterygoid muscle of one side acting in conjunction with the lateral pterygoid of the opposite side. On the side to which the jaw is moved the condyle remains almost stationary; on the opposite side the condyle and disc move downward and forward on the articular slope of the glenoid fossa as a protrusion.

The important superficial relations of the joint are the superficial temporal artery posteriorly, the highest branch of the facial nerve anteriorly, and the parotid gland laterally, posteriorly and inferiorly. On the deep aspect the maxillary artery runs forward medial to the neck of the condyle.

### Injuries

Injuries of joints due to acute mechanical violence may be subdivided into *closed* (or simple) injuries and *penetrating* wounds. Closed injuries may be further subdivided into *contusions*, *ligamentous injuries* and *internal derangements*.

Long continued or recurrent mild trauma leads to degenerative changes which will be described later in the chapter on diseases of joints (Chapter XX).

### Contusions

The synovial membrane may be contused by a blow over a joint. The membrane may also be torn in association with a sprain or dislocation.

The usual result of contusion is *traumatic synovitis*. The joint is painful and over a period of a few hours becomes distended with clear fluid. Movement is limited owing to the pain and the feeling of tension caused by the fluid.

Sometimes, in severe contusions, there is haemorrhage into the joint from torn vessels in the synovial membrane or as the result of an associated

intra-articular fracture. This condition, which is known as *haemarthrosis*, may also occur after trivial injuries—or even, apparently, without injury—in patients with haemophilia, scurvy or other haemorrhagic diseases.

Swelling due to haemarthrosis develops within an hour, and sometimes in a few minutes. If a large joint is affected the temperature usually rises by 2° or 3° F. X-ray examination may reveal an intra-articular fracture. These features usually suffice to distinguish the condition from traumatic synovitis, but in doubtful cases some fluid should be aspirated with a syringe and needle. If this is done every care must be taken to avoid introducing infection into the joint.

Traumatic synovitis is treated by applying a firm bandage and immobilizing the joint with a splint. Active movements of other joints are prescribed to prevent wasting of muscles. In traumatic synovitis of the knee, for instance, a splint is applied with the knee almost straight and the *quadriceps femoris* muscle is exercised by making the patient lie on his back and raise and lower the limb. Weight bearing is allowed in a day or two, and the splint can usually be discarded after about 10 days.

Blood in a joint often remains fluid but should clotting occur organization and subsequent permanent stiffness may ensue. In haemarthrosis, therefore, the blood should be aspirated, unless the patient is suffering from haemophilia when this might cause renewed and uncontrollable haemorrhage. After aspiration a pressure bandage is applied and the joint is immobilized. Exercises are postponed for a week or two to avoid re-starting the bleeding. The period of immobilization depends on the extent of the injury.

### **Contusion of the temporo-mandibular joint**

A blow on the jaw may cause traumatic synovitis of the temporo-mandibular joint. The main symptoms are pain and inability to open the mouth fully. The differential diagnosis from internal derangement will be discussed later.

The condition usually subsides in a few days if the mandible is immobilized in the position of rest.

### **Ligamentous Injuries**

Ligamentous injuries are subdivided according to the degree of damage sustained into sprains, subluxations and dislocations.

The term *sprain* is used when one (or more) of the ligaments of a joint is stretched or torn but the bones taking part in the articulation retain their normal relationship. *Subluxation* (sometimes called *partial dislocation*)



implies that the articular surfaces are partly separated as the result of severe damage to the capsular or accessory ligaments of the joint. *Dislocation* implies a still more severe grade of injury in which the articular surfaces are completely separated.

In addition to traumatic dislocation two other types must be recognized: *congenital dislocation*, which occurs usually in the hip joint, and *pathological dislocation*, which may occur in any joint and is the result of disease.

## **Sprains**

The joints most commonly affected are the knee and ankle.

### *Symptoms and signs*

The patient complains of pain in the region of the affected joint. On examination there is tenderness localized to the site of injury (usually near the point of attachment of the damaged ligament to the bone) and swelling of the subcutaneous tissues or the joint itself. The bones of the joint preserve their normal relationship and no abnormal mobility can be demonstrated.

Care is needed to distinguish a sprain from a subluxation which has undergone spontaneous reduction. In the latter condition, however, the subluxation can be reproduced by gentle manipulation and then recognized clinically or radiologically.

### *Treatment*

As a first aid measure a firm bandage is applied to limit swelling. Subsequently adhesive strapping or plaster of Paris is applied to protect the damaged ligament and, in the case of the ankle, the sole and heel of the boot is raised slightly on the side of the injury.

## **Dislocations**

We shall confine our attention to dislocations due to trauma.

### *Symptoms and signs*

Following injury the patient complains of pain in the region of the affected joint and some loss of function. On examination the following signs may be present:

1. Swelling in or around the joint.
2. Bruising in the tissues overlying the joint.
3. Deformity due to the abnormal relationship of the bones taking part in the articulation. This may be detected by observation and palpation, and confirmed by radiological examination. It is the most important sign of dislocation.

4. Limitation of the normal mobility of the joint.
5. Abnormal mobility. This means that it is possible to demonstrate movement of a kind which does not normally occur at the joint. The diagnosis is confirmed by X-ray examination (Fig. 28).



FIG. 28. Radiograph showing a sub-coracoid dislocation of the shoulder.

#### *Treatment*

The treatment of dislocations, like that of fractures, may be considered under three headings.

1. *Reduction*; that is, restoration of the normal relationship between the bones taking part in the joint. Reduction is usually effected by manipulation, and in recent dislocations anaesthesia is not absolutely essential

except in the case of the hip and elbow joints; if difficulty is experienced, however, an anaesthetic should be given. In dislocations of long standing operative reduction may be required.

As a general rule reduction should not be attempted until radiographs have been taken to determine the relationship of the bones and whether or not there is an associated fracture. If this would entail a long delay it is permissible to attempt reduction of a dislocation of a finger, the shoulder or the temporo-mandibular joint without waiting for radiological examination, *provided post-reduction films are taken at the earliest opportunity*. The attempt must be abandoned if difficulty is encountered.

2. *Prevention of recurrence*. Partial limitation of movement at the joint is usually sufficient to prevent recurrence. Thus, for example, a sling should be worn for two or three weeks after reduction of a dislocated shoulder. In the case of a weight-bearing joint fixation by splinting or plaster is required, and occasionally, following repeated dislocation of a joint, operative treatment may be necessary to prevent further recurrence.

3. *Restoration of normal mobility*. This is usually effected by graduated active movements. Occasionally, after prolonged immobilization, forcible manipulation of the joint (with or without an anaesthetic) is required.

The treatment of a fracture dislocation—that is, an injury in which there is dislocation of a joint and fracture of the articular end of one of the bones—follows similar lines, but operative reduction is often required and immobilization must be complete and prolonged.

### **Dislocation of the temporo-mandibular joint**

Dislocation of the temporo-mandibular joint may be unilateral or bilateral. In the great majority of cases the condyle of the mandible together with the articular disc is displaced forward on to or beyond the *eminentia articularis* of the temporal bone. The condition may result from a blow on the chin with the muscles of the jaw relaxed, from such acts as yawning or eating, from the use of a mouth gag or prop during dental or surgical operations when the capsule is unduly lax or the *eminentia articularis* is flat, or from the use of undue force in the extraction of teeth or roots, particularly if the jaw is not adequately supported and the patient has been given a general anaesthetic combined with a muscle relaxant. It may also occur during an epileptic fit.

On examination there is obvious deformity and loss of function. The jaw is held rigidly open, and the chin is projected forwards in bilateral cases and towards the uninjured side in unilateral cases. Speech and swallowing are seriously impaired and there is often profuse salivation.

Attempted movement causes severe pain. On palpation the head of the mandible is found to be displaced forward from its normal position.

Radiological examination is often considered unnecessary in recent cases, but is essential if there is any possibility of fracture-dislocation.

Treatment of recent uncomplicated dislocations is usually easy. The displacement is reduced by making downward pressure with the thumbs (suitably padded) on the posterior teeth (or on the alveoli in edentulous patients) and at the same time elevating the chin with the fingers outside the mouth. Anaesthesia is seldom necessary, but if reduction is not possible without it a general anaesthetic with a short acting muscle relaxant should be given. After reduction a supporting bandage should be applied for approximately a week and the patient should be maintained on a soft diet.

In neglected cases manual reduction may be impossible and an open reduction may be necessary, with subsequent repair of the capsule.

Recurrent dislocation of the temporo-mandibular joint is not very common but occurs in some people, often as a result of laxity in the capsule or anatomical abnormalities of the articular eminence and articular fossa. The condition is difficult to treat satisfactorily. Active exercises and muscle



FIG. 29. Radiograph showing a fracture dislocation of the right temporo-mandibular joint in a child.

re-education are important, and operations designed to tighten the capsule or increase the height of the articular eminences are undertaken in some cases. Some surgeons advocate in some cases injection of sclerosing agents into the periarticular tissues to produce fibrous adhesions around the joint.

In fracture dislocation—that is, fracture of the neck of the mandible and dislocation of the temporo-mandibular joint (Fig. 29)—reduction is difficult or impossible. In contrast to simple dislocation, however, there is usually little or no disturbance of the bite and a good functional result can often be obtained simply by maintaining the teeth in a position of normal occlusion by means of a firm supporting bandage or by fixation of the teeth themselves, and thereafter prescribing graduated exercises, with the use of a guiding flange if necessary. In most cases a false joint is formed. In children remoulding of the bone may occur to such an extent that after a few years no radiographic evidence of the injury remains (Fig. 30). Occasionally movement is poor or pain persists and condylectomy (i.e. removal of the condylar process) is then performed.



FIG. 30. Radiograph of the patient shown in Fig. 29 three years later. Reduction was not attempted but the deformity has disappeared as the result of bone growth and remoulding (Dr W.D. MacLennan's case).

### **Internal Derangements**

The term 'internal derangement of a joint' has been used in various senses since it was introduced by William Hey nearly a century and a half ago, but is best restricted to cases in which, as a result of injury or disease, some intra-articular structure has become displaced between the ends of the bones so that movement at the joint is restricted.

The limitation of movement is often spoken of as 'locking' but the term is misleading because it suggests complete fixation of the joint in one position and this rarely occurs in practice.

In addition to limitation of movement there may be audible 'clicking' when certain movements are attempted.

Internal derangement due to trauma occurs most frequently in the knee, as a result of injuries to the medial or lateral semilunar cartilages. It is also common in the temporo-mandibular joint and occurs sometimes in the sterno-clavicular joint.

Internal derangement due to disease may occur in any joint, and will be discussed further in the chapter on diseases of joints (Chapter XX).

#### **Internal derangement of the temporo-mandibular joint**

Internal derangement of the temporo-mandibular joint occurs in both males and females, most commonly in the late teens or early twenties.

The main etiological factors are partially or completely unerupted third molar teeth and irregularities in dental occlusion. The symptoms and signs include clicking, pain and limitation of movement.

These cases require careful investigation by a specialist including a detailed history, radiographs, study models and bite analysis.

Treatment is initially conservative. In cases due to mal-occlusion the first essential is to treat the underlying cause by such procedures as spot-grinding, removal of offending teeth or the fitting of an overlay splint. Some patients benefit from manipulation of the joint; in others pain may be relieved by the injection of long-acting local anaesthetic agents.

If conservative measures fail operation may have to be undertaken, the commonest indication for this being persistent severe pain. The procedures which are used include meniscectomy, subcondylar osteotomy ('condylectomy') and condylectomy.

## *Chapter VIII*

# HAEMORRHAGE AND SHOCK

### **Haemorrhage**

Haemorrhage is defined as the escape of blood from the vascular system. Omitting bleeding from the heart, which is rare, haemorrhage may be classified as *arterial*, *venous* or *capillary*. It is described as *external* when the bleeding is visible on the surface or at one of the orifices of the body, and as *internal* when bleeding occurs into the tissues, a hollow viscus or a serous cavity.

Haemorrhage is commonly due to trauma, but may occur spontaneously as a result of pathological changes in the vessel walls. Severe bleeding after quite trivial injury may occur in patients who are being treated with anti-coagulant drugs on account of arterial disease (Chapter XVII) or are suffering from haemorrhagic diseases.

### **The natural arrest of haemorrhage**

Haemorrhage may cease spontaneously as the result of three important reactions:

1. When blood comes in contact with damaged tissue, e.g. the wall of an injured vessel, blood platelets become adherent to the damaged tissue and to each other and, in addition, fibrin clot begins to form. Firm clotting causes temporary arrest of haemorrhage; organization of the clot causes permanent arrest.

2. When a large amount of blood has been lost the blood pressure falls and the tendency to bleeding is therefore reduced.

3. When an artery is completely divided the intima *retracts* and the media *contracts*, thereby tending to seal off the vessel (see Chapter XVII).

### **Mechanisms of platelet aggregation and blood clotting**

Platelet aggregation appears to be due to the release of a substance known as adenosine diphosphate (ADP) from the damaged tissue and probably also from the platelets themselves.

Blood clotting is an extremely complex process, culminating in the conversion of *fibrinogen* to *fibrin* by the enzyme *thrombin* and the subsequent stabilization of the fibrin in the presence of a fibrin stabilizing factor

(known as Factor XIII) and calcium. Thrombin is not present in detectable amounts in normal plasma (if it were the blood would clot); instead, plasma contains *prothrombin*, which is converted to thrombin when blood comes in contact with a foreign surface, or when enzymes from damaged tissues become mixed with it.

The conversion of prothrombin to thrombin is an extremely complex process.

When it is triggered off by contact with a foreign surface a substance in the plasma known as the Hageman Factor or Factor XII is activated and this sets up a chain reaction in which a series of substances which are present in normal plasma are activated in turn. These substances, in the order in which they are thought to be activated, are known as Factor XI, Factor IX, Factor VIII, and Factor X. Finally, activated Factor X, in the presence of another substance present in normal plasma known as Factor V, a phospholipid substance liberated by blood platelets, and probably also calcium ions, brings about the conversion of prothrombin to thrombin. The activation of Factor XII takes place relatively slowly; the subsequent reactions are faster.

The factors denoted above by Roman numerals in their activated form are probably all enzymes. It should be noted that the order in which they come into operation differs from their numerical order and that various numbers are missing from the series. The numbers I, II, III and IV do in fact denote respectively fibrinogen, prothrombin, tissue factor (see below) and calcium, but in practice the name is used in place of the number. There was at one time thought to be another factor which was given the number VI, but this appears to have been an error.

It may appear almost incredible that the process should be so complex, but according to R.G. Macfarlane, to whom a great deal of our understanding of the process is due, the system may be likened to a sort of six-stage amplifier, and appears to have evolved as a most efficient mechanism for converting the minute stimulus of surface contact into the sudden and powerful response of thrombin formation.

When material known as tissue factor from damaged tissue is mixed with blood the process is simpler, since tissue factor, in the presence of another substance present in normal plasma known as Factor VII, activates Factor X. Damaged tissues also liberate a phospholipid substance which has a similar action to that liberated by blood platelets so that the only additional substances needed to achieve the conversion of prothrombin to thrombin under these circumstances are Factor X, Factor VII, Factor V and probably calcium ions.



Clot formation is defective in patients receiving anticoagulants, and in those suffering from various haemorrhagic disorders including haemophilia, Christmas disease, von Willebrand's disease, severe liver damage, obstructive jaundice, thrombocytopenic purpura and some forms of leukaemia. There is a hereditary deficiency of Factor VIII (sometimes termed anti-haemophilic globulin) in haemophilia, and of Factor IX in Christmas disease. Liver disease may reduce prothrombin and Factors VII and X, as does the administration of anticoagulants of the dicoumarin type. Administration of heparin inhibits in a complex way the activation of prothrombin. In thrombocytopenic purpura the number of blood platelets is reduced. In von Willebrand's disease the bleeding time is prolonged and severe haemorrhage may occur from small vessels although the blood as a rule clots normally and there is no abnormal fibrinolytic activity. The disorder has been attributed to an abnormality of the capillaries but recent evidence suggests that there is a deficiency of some specific but as yet undefined plasma factor.

A certain amount of fibrin is constantly being formed as a consequence of unnoticed minor trauma to blood vessels, and pathological changes in the vascular endothelium (Chapter XVII). A mechanism exists for the elimination of unwanted clot by a process of enzymatic digestion termed *fibrinolysis*. The enzyme concerned, known as *plasmin*, is formed by the activation of an inert precursor *plasminogen*. Occasionally plasmin is formed in excessive amounts and abnormal bleeding may then occur because though clot is formed normally it is rapidly digested.

### **The restoration of blood lost**

Some blood may be restored to the circulation almost immediately by contraction of the spleen. In man (as distinct from some animals), however, the quantity is probably too small to be important.

Permanent restoration of the various constituents of the blood occurs in a definite sequence:

1. Fluid and electrolytes enter the circulation from the tissues. The rate at which this occurs varies, but after severe haemorrhage between 1 and 2 pints of fluid may enter the circulation within a few hours.
2. The plasma proteins are restored by the mobilization of protein from the tissues and by resynthesis. After haemorrhage of moderate severity the time required for this varies from a few hours to a few days.
3. The red blood cells and the leucocytes are gradually replaced over a period of several weeks as a result of proliferative activity of the bone marrow.

**The general symptoms and signs of haemorrhage**

Many patients who have lost blood have also sustained injuries of muscles, bones or viscera which complicate the clinical picture. For the moment, however, we are concerned with the effects of uncomplicated haemorrhage such as occurs when an artery is divided cleanly or eroded in the floor of an ulcer.

The general symptoms and signs of haemorrhage depend on the *amount* of blood lost, the *rate* at which it is lost and the *general condition of the patient* before the haemorrhage occurred. In normal subjects rapid loss of half a pint of blood produces a recognizable but slight effect; rapid loss of three pints is likely to be fatal unless blood transfusion is undertaken promptly. The symptoms and signs after fairly severe haemorrhage are as follows:

The patient feels weak and may faint; he often complains of intense thirst. On examination he is pale and often restless, and his extremities are cold and clammy. The temperature is usually subnormal. The pulse is rapid and has a peculiar quality; it is often described as 'collapsing' because the impulse, though it may be forceful initially, falls away rapidly. The blood pressure is low. Respiration is often rapid and gasping; it has been aptly described as 'air hunger respiration'. The volume of circulating blood is reduced but may be gradually restored by entrance of fluid from the tissues; when this occurs the haemoglobin concentration of the blood falls.

If a large amount of blood has been lost the patient becomes listless or even comatose, the pulse becomes scarcely perceptible, the respiration shallow and the blood pressure unrecordable. It is important to recognize and treat the condition promptly, because otherwise a patient whose life could have been saved may die before treatment is begun or pass into an irreversible state in which no treatment is of any avail.

**Local signs of external haemorrhage**

Haemorrhage may be arterial, venous or capillary. *Arterial haemorrhage* is recognized by the bright red colour of the blood and the fact that bleeding occurs in spurts synchronous with the heart beat. In *venous haemorrhage* the blood is dark red in colour and usually flows in a continuous stream. Occasionally spurting occurs from large veins in the neck, but the rhythm is respiratory not cardiac. In *capillary haemorrhage* the blood oozes slowly from a raw surface. Minor injuries sustained while shaving provide a familiar example.

Haemorrhage may also be classified as primary, reactionary and secondary. *Primary haemorrhage* occurs at the time of injury as the direct result of damage to vessels. *Reactionary haemorrhage* occurs up to about 24 hours after injury and is due to slipping of a ligature or displacement of a clot; it may develop as the blood pressure gradually returns to normal, or be precipitated by sudden increases in blood pressure due to coughing or straining. *Secondary haemorrhage* occurs usually about 10 days after a wound or operation and is due to infection causing softening and digestion of the wall of a vessel.

### **Treatment of haemorrhage**

The aim of treatment is twofold: to arrest bleeding and restore the various constituents of the blood.

As already described, both these objectives may sometimes be attained without active treatment. Spontaneous arrest of haemorrhage, however, is too uncertain to be relied on, and is rarely permanent except in trivial cases; and natural restoration of the constituents of the blood may not occur sufficiently rapidly.

### **Arrest of bleeding**

#### *Arterial haemorrhage*

In arterial haemorrhage due to a wound a firm dressing is applied as a first aid measure. If this is inadequate bleeding from a limb is controlled by applying a tourniquet or compressing the main artery against a bony point. If a doctor is present he should give an injection of morphia; this helps to diminish bleeding by allaying restlessness.

When the patient reaches hospital the wound is explored and, if possible, both ends of the divided vessel are ligated. In some situations this may be impracticable, and it may then be necessary to ligate the main artery to the part at a higher level. In severe bleeding from deep vessels in the hand, for example, it may be necessary to ligate the brachial artery, and occasionally the external carotid artery is ligated to control severe bleeding inside the mouth.

Primary arterial haemorrhage occurring during an operation is normally arrested by clamping and ligating the vessel concerned. With small vessels firm pressure may be sufficient, or electro-coagulation may be used.

#### *Venous haemorrhage*

Venous haemorrhage is usually arrested by firm pressure and, where possible, by elevation of the part. If a large vein has been completely

divided both ends must be ligated, but if only partly divided it may sometimes be repaired by lateral ligature, or by application of a muscle graft or a piece of gelatin sponge.

### *Capillary haemorrhage*

Capillary haemorrhage is easily arrested in normal people by firm pressure or by styptics of various kinds.

### *Haemorrhage following dental extraction*

After extraction of a tooth haemorrhage may occur from small vessels in the torn mucous membrane, from torn or stripped periosteum, or from the cancellous bone of the jaw. It is best controlled by inserting one or two sutures to approximate the lips of the socket.

## **Restoration of the blood—blood transfusion**

In mild cases it may be sufficient to give large quantities of *fluid by mouth*, followed later by a nourishing diet and a suitable preparation of iron to assist in the restoration of haemoglobin.

In more severe cases the ideal method of restoring the blood volume to normal is by *transfusion of whole blood* which is compatible with the blood of the recipient. In large centres blood is collected regularly from voluntary donors and stored in a blood bank, and it is the responsibility of those in charge of the bank to take every reasonable precaution to ensure that the donors are free of communicable disease, and to obtain the blood in an aseptic manner. In addition they undertake the blood grouping of donors, and usually also the grouping of recipients and the cross matching of donors and recipients. It is, however, the responsibility of the person giving the transfusion to see that only the correct blood is given; he should therefore be familiar with the basic principles of blood grouping, and in every case he must study the reports from the blood bank and check the label on the bottle.

### *The ABO system of blood grouping*

The existence of blood groups was discovered by Landsteiner in 1900 and within a few years the four primary blood groups were defined. These are designated as follows:

- Group AB or I
- Group A or II
- Group B or III
- Group O or IV

To determine an individual's blood group a sample of blood is taken and sodium citrate added to prevent coagulation. A drop of serum from an individual of group A (II) is placed on one slide and from an individual of group B (III) on another, and to each is added a drop of the unknown blood. The slides are gently rocked, and observed to see whether agglutination (clumping) of the added red cells occurs. The group of the unknown blood is then determined from the following scheme:

Cells agglutinated by group A serum only	Group B
Cells agglutinated by group B serum only	Group A
Cells agglutinated by both group A and group B serum	Group AB
Cells agglutinated by neither serum	Group O

As a general rule no incompatibility arises if the donor and recipient belong to the same blood group; or if, irrespective of the recipient's group, the donor belongs to group O (IV). For this reason members of Group O (IV) are called 'universal donors'. There are, however, exceptions to this rule and, except in cases of extreme urgency, direct 'cross-matching' should be undertaken as an additional precaution. To do this *donor red cells* are added to a drop of *recipient serum* on a slide, and if no agglutination occurs the transfusion may be given. Sometimes a test is also made using donor serum and recipient cells, but this is only necessary if the proposed recipient suffers from a blood disease or if repeated transfusions are to be given from the same donor.

*Subgroups of the ABO system.* Group A is now subdivided in  $A_1$ , and  $A_2$ , and group AB into  $A_1B$  and  $A_2B$ . Serum from people belonging to  $A_2$  occasionally agglutinates  $A_1$  red cells, and similarly serum from people of group  $A_2B$  may agglutinate  $A_1B$  cells, but so long as proper grouping and cross matching is undertaken no danger should arise on this account.

### *The Rh factor*

It was shown by Landsteiner and Wiener in 1940 that, in addition to belonging to one of the four primary blood groups, every individual may be classified according to whether his blood contains a factor known as the Rh or Rhesus factor. About 85 per cent of white people have this factor in their blood and are said to be 'Rh positive'; the remaining 15 per cent are 'Rh negative'. The subject of Rh grouping has become highly complex and Rh positive individuals are now further divided into a number of sub-types.

The discovery of the Rh factor has thrown light on a number of

unexplained transfusion reactions and on the etiology of haemorrhagic disease of the new born. The following points are of practical importance:

1. An Rh negative person may become sensitized to the Rh factor. This may result from a transfusion of Rh positive blood or may occur in a woman if she bears a foetus which is Rh positive.
2. A severe reaction may occur if Rh positive blood is transfused to an Rh negative recipient who has been sensitized to the Rh factor by previous transfusion or by pregnancy.
3. If a woman who is Rh negative becomes sensitized to the Rh factor and later bears an Rh positive foetus the child may be stillborn or may suffer from haemorrhagic disease of the new born.
4. It follows from the above that whenever possible Rh grouping should be undertaken before a transfusion is given, and an Rh negative patient should receive only Rh negative blood. If Rh grouping is not done the safe rule is to *give only Rh negative blood to any woman who is not too old to bear children or to any patient who has had previous transfusions.*

#### *Other grouping systems*

Many other blood grouping systems have been discovered, including the MNS, P, Lutheran, Kell, Lewis, Duffy and Kidd systems, and no doubt more will be discovered in the course of time. Differences between the donor and recipient in respect of the systems listed may be of importance if the recipient is suffering from certain disorders of the blood or has received a previous transfusion, and under such circumstances expert advice should be sought.

#### *Effects of transfusion of incompatible blood*

The patient usually complains first of *pain* in one or both loins. The pulse rate increases and the blood pressure may rise. The face becomes pale and the extremities cold due to constriction of vessels in the skin. Later signs include *flushing* as constriction of vessels in the skin gives place to dilatation, *rigor*, *difficulty in breathing*, *jaundice*, *haemoglobinuria*, and finally *oliguria* or *anuria*. (These last two terms denote diminished or absent secretion of urine respectively.) In some cases death occurs.

These effects occur because the incompatible red cells are agglutinated by the recipient's serum and subsequently haemolysed. Acid haematin is formed from the liberated haemoglobin and this substance blocks the tubules of the kidneys.

If any of the above symptoms and signs occur the transfusion should be stopped at once. In mild cases alkali is given by mouth in the form of

sodium citrate and sodium bicarbonate, but in more severe cases Ringer-lactate solution should be given by intravenous infusion. In all cases the blood remaining in the bottle and a sample of the patient's blood should be sent to the blood bank for investigation.

*Methods of transfusion. Blood substitutes*

Blood transfusion is illustrated in Figure 31, but the actual technique should be learned in the wards. The rate at which blood should be administered depends on the condition of the patient, but when the blood loss has been severe the first pint may be given in about 20 minutes and several pints may be given in 24 hours.

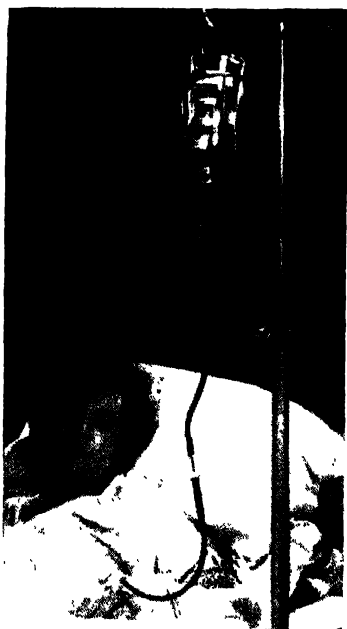


FIG. 31. Blood transfusion.

In an emergency, if whole blood is not available, plasma or one of the blood substitutes such as 'dextran' may be given. These materials do nothing to restore the oxygen carrying capacity of the blood, but they provide fluid, electrolytes and, in addition, substances of high molecular weight which, as explained in Chapter IX, help to prevent escape of fluid from the circulation to the tissues.

**General treatment**

In addition to the treatment described above *general treatment* is required; this includes rest, warmth and sedatives.

**Recognition and management of haemorrhagic disorders in surgical patients**

It is important to discover the existence of any tendency to abnormal bleeding before subjecting a patient to any form of surgical operation, including of course extraction of teeth.

All such patients should be asked routinely if they, or any member of their family, suffer from abnormal bleeding, and in all doubtful cases special tests should be performed. These include the platelet count, the bleeding time, the clotting time, the one-stage and two-stage prothrombin estimations and the level of antihaemophilic globulin in the plasma.

If there is a definite defect in the clotting mechanism appropriate treatment must be given preoperatively. Vitamin K reverses the effect of anticoagulants of the dicoumarin type, and is of value also in patients with obstructive jaundice and liver disease. Ordinary bank blood can be used to make good a deficiency of Factors VII, IX, X and prothrombin; fresh blood provides also Factor V and blood platelets. Excessive fibrinolysis may be corrected by administration of  $\Sigma$ -aminocaproic acid (EACA).

In mild haemophilia fresh whole blood may suffice to cover a small operation, but in severe cases injections of antihaemophilic globulin (AHG) are given. Ideally, human AHG is used but the supply is limited and it may be necessary to use AHG from animals. This is readily available but has the disadvantage that, being a foreign protein, it is antigenic. In consequence it usually cannot be given for more than 10–14 days because it ceases to be effective and may precipitate allergic reactions.

**Shock**

There is no precise or generally accepted definition of shock, but the term is used clinically to denote a state of circulatory collapse resulting from a variety of causes including fright, wounds, burns and acute perforations of hollow abdominal viscera. The use of a single word in this way has been the cause of much confusion; it suggests that we are dealing with a single pathological entity when, in fact, we are dealing with a variety of entities which differ in their pathogenesis and clinical features. We shall, therefore, avoid using the word 'shock' without qualification, but will continue to speak of 'wound shock', 'shock due to burns' and so on. In this chapter we shall confine our attention to wound shock.

**Wound shock**

Severe wound shock is almost always associated with reduction in the volume of *circulating* blood. This condition, which is termed *oligaemia* or



*hypovolaemia*, may result from escape of fluid from the vascular system or stagnation of blood in dilated capillaries. The part played by each of these factors has been much disputed but investigations during the second world war have proved that in wound shock the most important cause of the oligæmia is hæmorrhage; there are additional subsidiary causes but the mechanism of these is not fully understood. It follows that the clinical features of oligæmic wound shock and hæmorrhage are identical, and attempts to differentiate between these conditions are based on a misconception. Prompt and adequate blood transfusion is the essential treatment.

There is another type of wound shock which may follow any injury, serious or trivial; it is sometimes precipitated by minor medical or dental procedures, including injections. This condition is rarely serious and is not associated with oligæmia. It is often referred to as *primary shock* because symptoms develop immediately after injury; alternatively, it is called *neurogenic shock* because it appears to be due either to psychic factors or to painful impulses transmitted by the sensory nerves from the injured area. The vasomotor and cardiac centres in the medulla are disturbed and as a result there is vasodilatation causing a fall in blood pressure, and slowing of the heart due to impulses transmitted along the vagi. The vasodilatation affects mainly the arterioles in skeletal muscle; it does not occur in the vessels of the skin.

The patient feels suddenly faint. His face is pale and his extremities are cold and clammy. The pulse is *slow* and feeble, the respirations shallow and the blood pressure low. A similar condition occurs in some subjects without apparent cause.

The patient should lie down with his head low; he should be reassured and kept warm. Recovery is usually rapid, but if the blood pressure remains low 15 milligrammes of *methedrine* (a vasoconstrictor drug) may be given intravenously.

### **The Crush Syndrome**

The crush syndrome is another result of injury which attracted attention during the first world war and was much investigated during the second. It usually follows prolonged application of a tourniquet, or a limb injury in which muscles are severely crushed. The limb becomes swollen and paralysed, and the main artery goes into spasm. There is usually a fall in blood pressure, and renal failure, with oliguria or anuria, develops within a few days.

The damage to the kidneys has been variously attributed to the toxic effect of products of muscle breakdown and to blocking of the renal tubules by myohaemoglobin.

As a prophylactic measure an irretrievably damaged limb should be amputated without undue delay. If renal failure develops the intake of fluid and protein is restricted, and resonium enemas are given to reduce the level of serum potassium if this rises unduly. In severe cases, the patient may require peritoneal dialysis, or haemodialysis with the artificial kidney.

## *Chapter IX*

# INJURIES DUE TO

# THERMAL AND CHEMICAL AGENTS

### **Burns and Scalds**

Injuries caused by heat are called *burns*. This term is also used for similar injuries caused by electric currents, X-rays, radium and ultra-violet light; we shall, however, consider only thermal burns. Burns caused by moist heat—for instance, by boiling water—are often referred to as *scalds*. Intense heat causes actual charring of tissues; heat insufficient to cause charring may kill cells by destroying their enzymes and coagulating their protein.

Burns are classified as *superficial* or *deep*. In a superficial burn there is only partial skin loss; in a deep burn the whole thickness of the skin is destroyed. A patient may, of course, have superficial burns in some areas and deep burns in others.

### **Result of burns**

The important sequelae of a severe burn are shock, infection, persistent ulceration and deformity. These may be minimized by adequate treatment.

#### *Burn shock*

Immediately following injury there is often a stage of primary shock similar to that which follows wounds. This is usually far less serious than the delayed or secondary shock which is liable to supervene some hours later.

Secondary burn shock, like severe wound shock, is associated with oligæmia (Chapter VIII). In burn shock, however, the fluid lost is blood plasma, not whole blood. The plasma escapes from damaged and dilated capillaries at the site of the burn; it may ooze from raw surfaces, collect in blisters or be retained in the damaged tissues where it gives rise to local *oedema*. Loss of plasma causes a fall in the total volume of circulating blood and an increase in the ratio of the volume of cells to the volume of plasma in the circulation. Increase in this ratio is termed *haemoconcentration*; it is accompanied by increase in the red cell count and in the concentration of haemoglobin in the blood. Haemoconcentration may be demonstrated directly by centrifuging a sample of blood in a graduated tube known as a *haematocrit*. In deep burns the increase in red

cell count due to haemoconcentration may be partly counterbalanced by destruction of red cells (haemolysis) in the burned area. If there is much haemolysis a sample of plasma appears red in colour.

Is loss of plasma the only factor in burn shock? Until recently burn shock was ascribed to the action of toxic substances liberated by the injured tissues, but when the importance of plasma loss came to be realized this theory was largely abandoned. Toxaemia does appear to play a part in some cases, however, since a patient may die some days after a severe burn despite replacement of the lost plasma and control of infection.

The degree of shock resulting from a burn depends mainly on the *area* of skin affected. In extensive burns shock is often the cause of death.

#### *Bacterial infection of the burned area*

Infection of the burned area may have disastrous results. Bacterial toxaemia or septicaemia (Chapter X) may prove fatal. Apart from these general effects infection may enhance the local tissue destruction so that a partial-thickness skin loss becomes converted into whole-thickness loss and the resulting scarring is greatly increased.

#### *Persisting ulceration*

After a superficial burn healing occurs once the infection has been overcome and dead tissue has separated. Granulation tissue forms and gradually becomes transformed into fibrous tissue, and epithelium grows over the surface, partly from the periphery and partly from islands of epithelial tissue in the floor of the burn.

In deep burns there is no surviving epithelium in the floor of the burn. If the area is large ingrowth of epithelium from the periphery is insufficient to cover it and an ulcer (Chapter XIV) remains which will need skin grafting.

#### *Deformity*

Deformity may result from the contraction of scar tissue especially after deep burns. In addition to being unsightly this may entail serious disability. A burn over the flexor surface of a joint, for instance, may be followed by permanent limitation of extension. A burn below the eye may cause eversion of the lower lid (*ectropion*), and one on the side of the face may lead to limitation of movement of the jaw. Burns of the hands commonly result in stiff fingers.

### Treatment of burns

The principles to be observed in the treatment of burns correspond to the sequelae described in the last section. They are as follows:

#### 1. *Prevention and treatment of shock*

Burn shock, as we have seen, is due mainly to loss of plasma from the circulation; the object of treatment must therefore be to prevent this loss or to restore the blood volume by administration of fluids.

*Attempts to prevent plasma loss.* The first step was the introduction of the tannic acid treatment in 1925. Tannic acid (2 per cent to 5 per cent solution) was sprayed on the burned area and by coagulating the serum exuding from the burn limited the absorption of toxic products and reduced fluid loss. This method has three grave disadvantages: (a) sepsis often develops beneath the coagulum; (b) the coagulum has a constricting effect which, on the limbs, may seriously interfere with the blood supply to the part and cause gangrene; (c) tannic acid is a toxic substance and sufficient may be absorbed to cause severe liver damage. The first two of these disadvantages apply also to other coagulants which have been tried, and this form of treatment has therefore been abandoned.

*Restoration of blood volume.* Replacement of fluid by intravenous infusion is now undertaken in every patient with severe burns. Intravenous saline is not very effective because the extra fluid soon escapes from the circulation into the tissues, but plasma and whole blood are effective by virtue of their high protein content. The protein molecules, being large, do not escape so easily, and by their osmotic effect limit the escape of water. Initially plasma is given; later, if the patient becomes anaemic owing to haemolysis or superadded infection, whole blood is used. If neither plasma nor whole blood is available dextran solution is useful as a substitute.

Plasma is administered in the same way as whole blood, but grouping is not required. It is very rare for any immediate unfavourable reaction to occur, but sometimes *jaundice* develops months after the infusion. This appears to be due to a filtrable virus carried in the plasma. It is said not to occur if the plasma has been stored for some weeks at *room temperature* instead of in the refrigerator.

The need for giving plasma and the quantity required depend on the extent of the burn and the age of the patient. In adults infusion is usually necessary if more than 12 per cent of the body surface is burned; children are more susceptible to shock and need infusion for less extensive burns.

In addition to replacing lost fluid the patient must be kept warm and given morphia or other drugs for the relief of pain.

## *2. Prevention of sepsis*

The burned area is cleansed as soon as possible after injury with one of the modern detergents such as cetrimide. A non-adherent dressing is then applied, or the area is dusted with penicillin powder and left exposed. Alternatively, a burned limb may be enclosed in a watertight bag (the Bunyan-Stannard bag) which is continuously irrigated with warm saline.

Occasionally, in deep burns, if the patient's general condition permits, dead tissue may be excised immediately under anaesthesia and skin grafts applied; usually, however, this is best postponed for about 10 days.

Antibiotics are given in all cases. A combination of penicillin and streptomycin by injection may be used initially, but swabs from the burned area should be sent frequently for bacteriological culture and other antibiotics substituted as required according to the sensitivities of the predominant organisms.

## *3. Promotion of healing and prevention of deformity*

In deep burns skin grafting is necessary to obtain healing. This is usually undertaken about 10 days after burning. Early skin grafting helps to prevent scarring and consequent deformity, but some delay is usually necessary on account of shock and bacterial infection. Once grafts have taken gentle active exercises should be started; prior to this the part should be splinted in the position of optimum function.

If deformity has already developed the only satisfactory treatment is to excise the scar and apply a skin graft.

## **Injuries due to Chemical Agents**

Strictly speaking every case of poisoning is an example of injury due to a chemical agent, but here we shall confine our attention to the effects of corrosive substances such as strong acids or alkalis, vesicant gases and lysol. These effects depend on whether the corrosive is spilled on the skin or taken internally.

### **Corrosives applied to the skin**

Corrosive substances coming in contact with the skin produce lesions closely resembling burns. The immediate treatment is to wash off or neutralize all traces of the corrosive as quickly as possible. If the lesions are due to mustard gas or lewisite, injections of BAL (British anti-lewisite) are given. Subsequent treatment is similar to that described for burns. Healing is often exceedingly slow.

**Corrosives taken internally**

Lesions occur in the mouth and throat, and, if the substance is swallowed, in the oesophagus and stomach. The common sequelae are shock, infection, scarring with the formation of a stricture (Chapter XXIII), and in some cases, perforation.

Treatment is difficult. The mouth and throat are washed out, using an alkaline solution if the corrosive was acid and vice versa. If the corrosive has been swallowed a stomach tube is passed and the stomach also is washed out. Anti-shock treatment is instituted and antibiotics are given systemically as prophylaxis against infection. In the later stages various operative procedures may be necessary on account of stricture formation.

**Skin Grafting**

Reference has been made in this chapter to the use of skin grafting, and there are certain general facts about this procedure which we must now briefly consider.

Skin grafts are of two main kinds: free grafts and flaps. A free graft is completely detached before being transferred to its new site; a flap, on the other hand, remains connected to its site of origin, at least for a time, by a pedicle containing blood vessels.

**Free grafts**

Only autografts (that is, grafts of the patient's own skin) survive permanently. Grafts from other individuals may survive for a week or two but are then destroyed.

The following types of graft are in common use:

1. *'Pinch' grafts.* These consist of pieces of skin about  $\frac{1}{4}$  inch in diameter which are thin at the periphery and almost full thickness in the centre. They are obtained by lifting up a small area of skin with the point of a needle and slicing it off with a sharp knife, and are either laid on the area to be grafted or buried in the granulation tissue.

Pinch grafts are easy to cut, and often take well even when there is appreciable infection of the recipient area, but they do not yield a very good cosmetic result.

2. *Split skin or Thiersch grafts.* These are cut freehand with a skin-grafting knife, or with a mechanical apparatus known as a dermatome. They are of varying degrees of thickness and include the epidermis and part of the dermis. Split skin grafts may be applied in large sheets or cut into strips or small squares (Fig. 32). They take well in clean areas and yield good results in parts not subjected to pressure.

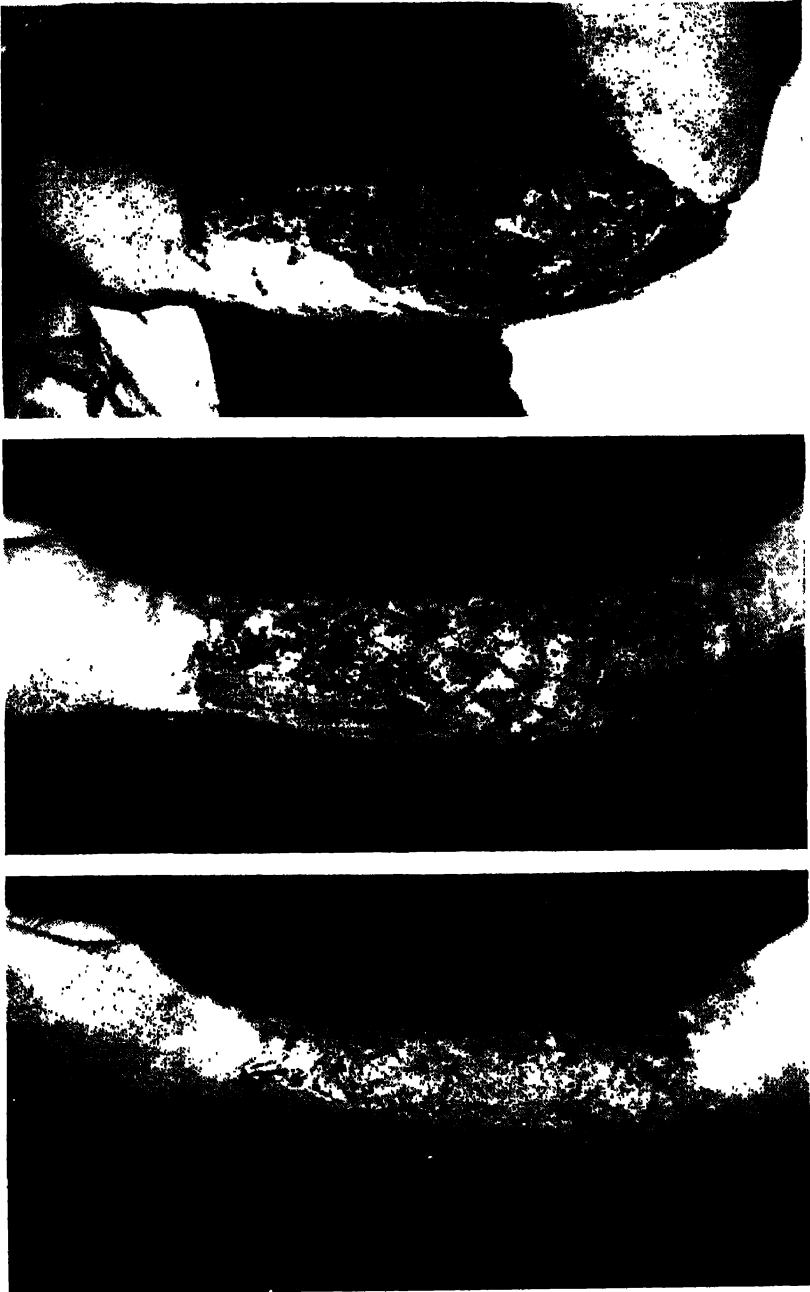


FIG. 32. Split-skin grafts applied in the form of small squares for the treatment of deep burns.

*A.* Before grafting.

*B.* A few days after grafting.

*C.* Two weeks after grafting showing coalescence of epithelium.

(Mr W. Hynes' case)



Though most commonly used to replace lost skin, split skin grafts may be used for repairing mucous membranes in the mouth and elsewhere. For this purpose they are usually applied on moulds of dental wax.

3. *Whole thickness grafts.* These take only on fresh raw surfaces which are free from infection and have a good blood supply. When successful they give an excellent cosmetic result.

### **Flaps**

A flap consists of skin and subcutaneous tissue. Originally its blood supply is derived from vessels entering the pedicle; after a time, however, vessels grow in from the new site and when these are well developed the pedicle may be divided.

Flap grafting is often a complex procedure but is necessary when full-thickness cover is required and conditions are unsuitable for free thick grafts.

## *Chapter X*

# PYOGENIC BACTERIAL INFECTION

Bacteria which are able to cause suppuration are termed *pyogenic*. They include staphylococci, streptococci, gonococci, *B. coli* and other less common organisms. It is important to realize that while some pyogenic bacteria such as staphylococci habitually cause suppuration, others do so only under special circumstances.

In this chapter we shall discuss some common examples of pyogenic bacterial infection under the following headings:

1. Acute abscess.
2. Cellulitis.
3. Pyogenic infection of lymphatics and lymph nodes.
4. Pyogenic infection of mucous surfaces.
5. Focal sepsis.

Pyogenic infection of bones and joints will be considered in Chapters XIX and XX respectively.

### **Acute Abscess**

An abscess is a collection of pus, surrounded by a zone of inflamed tissue and sometimes containing a mass of dead tissue known as a *slough*. The lesion known as 'cold abscess', which occurs in tuberculous infections, is not covered by this definition and is not a true abscess.

True abscesses (with rare exceptions such as amoebic abscess) are caused by infection with pyogenic bacteria. The majority are acute, but chronic abscesses sometimes occur, especially in bone or around retained foreign bodies. In this section we shall consider only acute abscesses.

The organisms responsible may reach the area in one of two ways:

1. From the surface of the body—through a wound, a small (perhaps microscopic) abrasion, a hair follicle or the orifice of a small gland.
2. From a focus of infection elsewhere in the body—usually via the blood stream, the lymphatic system or a serous cavity.

Usually the abscess develops soon after infection has taken place, but occasionally organisms lie dormant in the tissues for years and suddenly become active as a result of injury to the part.

The organisms form toxins which cause *necrosis* (that is, death) of nearby tissues. The dead material is gradually separated from the surrounding living tissue by the action of phagocytic cells, especially polymorphonuclear leucocytes. It may remain as a slough or may be broken down, partly by *autolysis* (digestion by its own enzymes) and partly by the action of the polymorphs which, besides being phagocytic, secrete a powerful proteolytic enzyme.

The pus consists of polymorphs and other inflammatory cells (most of them dead), products of tissue breakdown, bacteria and their metabolic products, and blood serum exuded from the vessels.

### **Symptoms and signs**

#### *1. Local symptoms and signs*

The usual symptoms and signs of acute inflammation described in Chapter IV are present. In addition, if the abscess is superficial, fluctuation may be demonstrable; if it is deeply situated there may be brawny oedema of the overlying tissues.

#### *2. Symptoms and signs due to pressure on neighbouring structures*

Symptoms and signs due to pressure on neighbouring structures depend, naturally, on the situation of the abscess. A common example is pain due to pressure on nearby nerves or nerve endings.

#### *3. General symptoms and signs*

The general symptoms and signs depend largely on the size and site of the abscess, and on whether or not drainage has been established.

In some cases there is only mild pyrexia (for example 99° F.), with a correspondingly slight increase in pulse rate; in others there is a high swinging temperature (for example, to 104° F.), a rapid pulse, loss of appetite, headache, vomiting and constipation.

Sugar may be present in the urine for one of two reasons:

- (1) Suppuration occurs very commonly in diabetics.
- (2) Even in non-diabetics glycosuria may occur as a result of septic infection.

In order to be sure of the significance of glycosuria in a patient with an abscess it may therefore be necessary to estimate the fasting blood sugar and perhaps also to perform a glucose tolerance test.

### Course and treatment

When only a small amount of pus is formed, and the slough is sufficiently small to be broken down completely, absorption may occur and the lesion may heal spontaneously.

This process may be expedited by the various forms of conservative treatment of acute inflammation described in Chapter IV.

In more serious cases the pus gradually increases in amount and the tension in the abscess rises, causing severe pain, and constitutional symptoms due to the absorption of toxic products. Spontaneous healing may still occur if the abscess is sufficiently superficial for the pus to burst through to the surface, but it is rarely justifiable to wait for this to happen. *Pus under tension should be evacuated by surgical means as soon as the diagnosis is made.* Local anaesthesia is contra-indicated as it may cause spread of infection so some form of general anaesthetic is normally used; occasionally, in the case of minor lesions, the part is frozen by means of an ethyl chloride spray.

Abscesses are usually best opened by Hilton's method. A small incision is made at the centre of the fluctuant area, or, if fluctuation is not present, over the site of maximal tenderness; a sinus forceps is then inserted and when pus is found the blades are widely separated. Sometimes a piece of rubber drain is then inserted and left in place for a few days.

An incision for opening an abscess (or any other purpose) on the face or neck should, whenever possible, be made in one of the skin creases. Such incisions heal well and leave inconspicuous scars; incisions in other directions tend to gape and leave broad ugly scars.

### Boils and carbuncles

A *boil* is a small subcutaneous abscess caused by infection of a hair follicle. It contains a slough known to the laity as the 'core'. Conservative treatment is usually adequate, but sometimes incision is required.

A boil on the upper lip is especially dangerous as infection may spread via the angular vein to the cavernous sinus; this complication may prove fatal. The patient should therefore be put to bed and treated by the application of a sterile dressing and by administration of penicillin. A sample of pus is sent for culture and if the organisms are found to be penicillin-resistant another antibiotic is substituted (see Chapter XI). Incision or 'squeezing' must be rigorously avoided.

A *carbuncle* (Fig. 33) is caused by pyogenic infection of subcutaneous tissue. There is usually extensive necrosis but relatively little pus formation,

so that the lesion may be regarded as an area of infective gangrene (Chapter XVI) rather than an abscess.

Treatment is usually conservative and should include injections of penicillin. The remarks made with reference to a boil on the upper lip apply even more strongly to a carbuncle in this region.

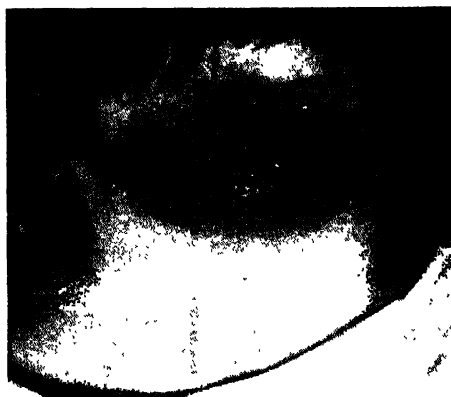


FIG. 33. A carbuncle on the back of the neck.

### **Alveolar abscess**

Apical infection, associated with a carious or dead tooth or a retained root, may spread through the bone to form an abscess in the soft tissue of the gum. This complication, which is known as alveolar abscess, is likely to occur if warning dental symptoms are ignored or if they are masked because the affected tooth is dead.

From the mode of spread one might expect infection of the bone (osteomyelitis) to accompany every alveolar abscess; fortunately, however, it does so only occasionally.

*Treatment.* The offending tooth or root must be extracted. In addition to removing the cause this may provide effective drainage of the abscess, especially if the lesion is situated in the upper jaw. In the lower jaw dependent drainage cannot be achieved in this way and incision through the gum or externally is often needed. Frequent hot mouth washes are ordered and penicillin or some other appropriate antibiotic is given in severe cases to help to control infection. If induration persists after the acute pain has subsided infra-red radiation is of value.

### **Peritonsillar abscess**

A peritonsillar abscess or 'quinsy' sometimes occurs as a complication of acute tonsillitis (page 126). The patient complains of severe pain and difficulty in swallowing. On examination there is a tense bulging swelling

extending downwards from the soft palate (Fig. 8); this is usually seen on one side only, but occasionally the condition is bilateral. The treatment is to evacuate the pus promptly by thrusting a sinus forceps into the centre of the swelling. This is often done without anaesthesia, but local application of xylocaine makes the procedure less painful. The patient should be sitting up and should lean forward as soon as the pus is evacuated. General anaesthesia should not be used as there would be great danger of aspiration of pus.

### **Retropharyngeal abscess**

In children suppuration sometimes develops between the posterior pharyngeal wall and the prevertebral fascia; the pus remains on one or other side and is prevented from spreading across the mid-line by a firm fibrous septum. This condition, which is known as acute retropharyngeal abscess, develops usually as a complication of one of the infectious fevers. The treatment is to evacuate the pus in the manner described for quinsy.

Acute retropharyngeal abscess must be distinguished from a retropharyngeal 'cold abscess' (Chapter XIII) resulting from tuberculosis of the cervical spine. Here the swelling is posterior to the prevertebral fascia and extends freely across the mid-line. It would be a surgical crime to open a cold abscess into the pharynx.

### **Cellulitis**

Cellulitis is defined as acute inflammation in cellular tissue. It must be realized that the term 'cellular tissue' refers to loose connective tissue, such as the subcutaneous tissue, which is cellular in the sense that a honeycomb is cellular; it does not, as one might expect, mean tissue rich in cells.

The causal organism is nearly always a streptococcus and the portal of entry is usually a wound or abrasion.

In mild cases resolution may occur, but often the condition extends widely in the subcutaneous tissues or via the lymphatics, and suppuration develops. Septicaemia sometimes follows.

Clinically there is a red, hot, brawny, tender, oedematous area, in the centre of which fluctuation may or may not be demonstrable. The related lymph nodes are usually enlarged and tender, and the general symptoms and signs of inflammation may be severe.

Treatment follows the usual lines of rest, heat, elevation and administration of an appropriate antibiotic. If frank suppuration occurs one or more incisions must be made to evacuate the pus.

Cellulitis on the face is particularly dangerous because of the risk of spread to the cerebral venous sinuses via the angular vein or the pterygoid plexus.

### ✓ **Ludwig's angina**

Ludwig's angina is a condition in which there is acute cellulitis of the floor of the mouth and the upper part of the neck, including the submental and submaxillary triangles. There is marked oedema and induration. It is unusual to find a large collection of pus, but small foci of suppuration may be scattered through the affected tissues.

The causal organism is usually a haemolytic streptococcus; sometimes, however, staphylococci or pneumococci are present, either alone or in combination with other organisms. Infection apparently enters through a trivial lesion in the mouth.

There are two main dangers: inflammatory oedema of the glottis causing respiratory obstruction, and spread of infection to the mediastinum. Prior to the discovery of sulphonamides and antibiotics the mortality was over 50 per cent.

*Treatment.* An incision should be made in the brawny area of the neck at the earliest possible moment; this lowers the tension even if not much pus is found. Gas and oxygen is the anaesthetic of choice; *intravenous anaesthetics such as thiopentone are contra-indicated* since they are likely to precipitate glottic spasm. If respiratory obstruction develops an endotracheal tube should be passed if possible; failing this, emergency tracheostomy offers the only hope, but septic pneumonia is likely to follow.

A sample of the pus, or failing this a swab from the tissue exposed at operation, is sent for culture. While awaiting the result the patient is given full doses of penicillin because the organisms are usually penicillin sensitive. If however they are found to be resistant another antibiotic is substituted.

### **Suppuration in the scalp**

*Superficial suppuration in the scalp* sometimes occurs in children as a complication of *pediculosis capitis*. Abrasions are produced by scratching and readily become infected. Acute lymphadenitis is a common sequel.

The pediculi are nowadays disposed of by applying preparations containing DDT. If serious infection has occurred it may be necessary to shave the whole head.

*Subgaleal suppuration* may result from a wound or from secondary infection of a haematoma. Treatment is in accordance with general principles; namely, free drainage by one or more incisions, combined with chemotherapy or administration of antibiotics.

*Suppuration deep to the pericranium* is usually a consequence of osteomyelitis of the skull. It is a serious condition as infection may spread to the meninges.

### **Pyogenic Infection of Lymphatics and Lymph Nodes**

#### **Lymphangitis**

Lymphangitis means inflammation of lymphatic vessels. It is almost always due to streptococci and occurs usually on the extremities as a complication of a small wound or abrasion. On inspection the inflamed lymphatics are seen as red longitudinal streaks, and on palpation they are felt as indurated cords. There is often high fever and sometimes a rigor; septicaemia may occur if the condition is not properly treated.

The treatment is to put the limb at rest, deal appropriately with the wound if such be present, and give an appropriate antibiotic. The causal organism is usually a haemolytic streptococcus, sensitive to penicillin.

#### **Lymphadenitis**

Lymphadenitis means inflammation of lymph nodes. It may be acute or chronic.

Acute lymphadenitis is usually due to streptococci or staphylococci. It occurs in the nodes related to a focus of infection, commonly a small wound or abrasion, but by the time the condition is well developed this focus may have healed. The nodes are enlarged and tender. Suppuration may or may not occur; if it does an abscess is formed.

The treatment is to put the part at rest, treat the focus from which organisms reached the node and, in severe cases, give an appropriate antibiotic. If an abscess forms it will require incision.

#### **Erysipelas**

Acute streptococcal infection of the lymphatics of the skin is known as erysipelas. A similar condition occurs occasionally on mucous membranes. Erysipelas is commoner in women than in men. It may occur as a complication of a wound or abrasion, but usually arises in the absence of any such lesion. Any part of the body may be affected, but the commonest site is the face.

Locally there is a bright red rash with a raised edge which spreads centrifugally as the centre of the patch clears. The skin is stiff and painful. Vesiculation is common but suppuration is very rare. The related lymph nodes are usually enlarged and tender.



The constitutional symptoms vary from mild pyrexia to severe fever with a rapid bounding pulse, rigors and sometimes delirium.

At one time erysipelas was a common and serious condition, with an appreciable mortality especially in infants and old people, but the outlook was dramatically improved by the development of the sulphonamides. Today erysipelas is something of a rarity. If seen it is best treated with penicillin, to which it responds promptly.

Erysipelas is readily transmitted by contact so that the patient should be isolated, and doctors and nurses treating the case should take appropriate precautions to avoid carrying the infection on hands or clothing.

### **Pyogenic Infection of Mucous Surfaces**

Inflammation on mucous surfaces may be due to a wide variety of organisms, including bacteria proper and filtrable viruses. Mixed infections are common. In the present chapter we are concerned only with inflammation due solely or mainly to pyogenic bacteria.

In mild cases inflammation on a mucous surface may resolve promptly despite the presence of pyogenic organisms. Failing this the following complications may develop:

1. *Formation of an empyema.* When the inflammation occurs in the mucosa lining a cavity such as the maxillary sinus or the gall-bladder, a collection of pus, sometimes under considerable tension, may develop in the cavity itself. This is called an empyema.

The term empyema is also applied to a collection of pus in a pleural cavity, though the pleura is lined by endothelium and not mucous membrane.

2. *Formation of a submucous abscess.*

3. *Diffuse suppuration in the submucosa with extensive sloughing of the mucosa.* When this occurs the condition is sometimes known as *phlegmonous inflammation*.

4. *Healing with stricture formation.* When inflammation of the mucous membrane lining a narrow tube extends into the submucosa there may be subsequent localized narrowing of the tube due to the contraction of scar tissue. The narrowed region is termed a *stricture*. Stricture of the urethra was a common complication of acute gonococcal urethritis before the discovery of the sulphonamides and penicillin.

We shall illustrate pyogenic inflammation on mucous surfaces by describing some common inflammatory conditions of the nose, ear and throat.

**Suppuration in the nose and accessory nasal sinuses***Surgical anatomy*

The nasal cavity lies between the base of the skull and the roof of the mouth, and is divided by the nasal septum into right and left nasal fossae. The upper third of the inner and outer walls of each nasal fossa is lined by specialized olfactory epithelium, the remainder by ciliated columnar epithelium.

On the lateral wall of each fossa are the superior, middle and inferior turbinate bones; these partly divide the fossa into the superior, middle and inferior meatuses (Fig. 34).

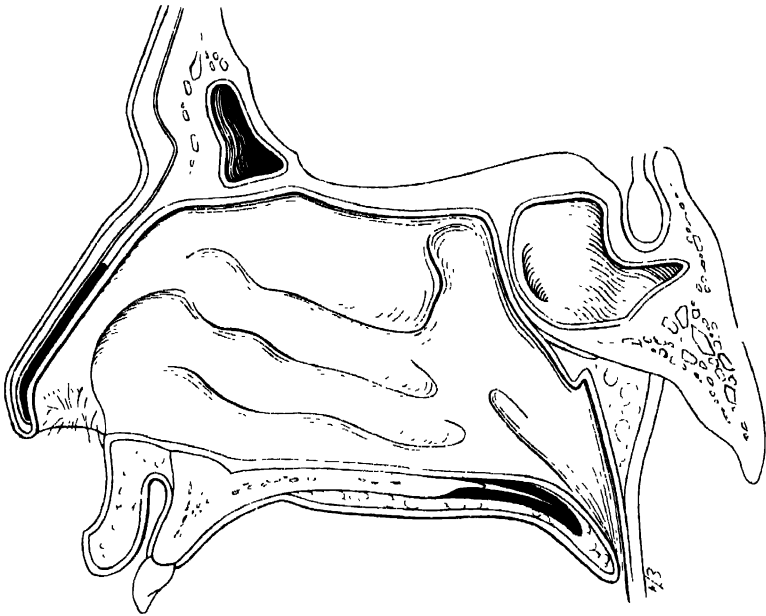


FIG. 34. The lateral wall of the nose.

The accessory nasal sinuses are cavities lined by ciliated columnar epithelium which is continuous with the epithelium of the nasal fossae. They comprise on each side the sphenoidal, frontal and maxillary sinuses and the ethmoidal air cells.

The sphenoidal sinuses (Fig. 34) lie within the body of the sphenoid bone, immediately below the pituitary fossa. They are separated from each other by a thin plate of bone, and each opens into a small space known as the sphenothmoidal recess, situated above and behind the corresponding superior turbinate bone.

The frontal sinuses (Fig. 34) are pyramidal shaped spaces which develop in the frontal bones about the seventh year of life. The extent to which they develop is very variable and one or both sinuses may be absent. Each opens to the anterior part of the middle meatus on the corresponding side.

The maxillary sinuses—sometimes called antra—are the largest of the sinuses. Each is a pyramidal shaped space which occupies most of the corresponding maxilla (Fig. 35). The apex of the pyramid lies in the zygomatic process of the maxilla; the superior wall is the orbital plate which

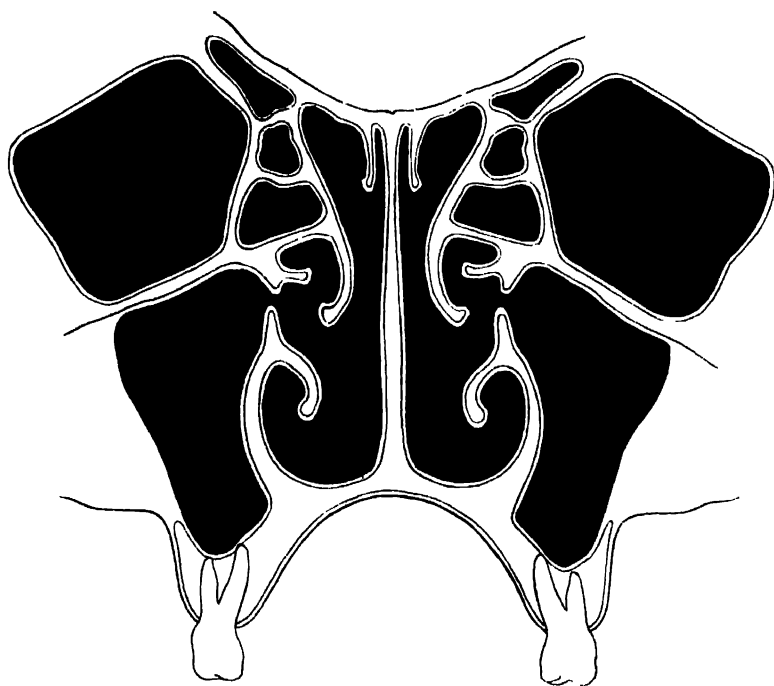


FIG. 35. Coronal section through the nose, antrum and orbits.

separates the sinus from the infraorbital nerve and the contents of the orbit; the anterior wall looks towards the face and is very thin in the region of the canine fossa. The inferior wall or floor is formed by part of the alveolar border of the maxilla. The number of teeth whose roots are related to the floor of the sinus is variable. Often only the molars or the molars and the second premolar are so related, but the sinus may extend so far forward as to be related to all the teeth except the incisors. The roots of the first and second molars sometimes project a considerable distance into the sinus, being separated from the actual cavity only by a very thin

layer of bone and mucous membrane. The medial wall separates the sinus from the nasal cavity; high up on this wall there is an opening through which the sinus communicates with the middle meatus.

*Acute sinusitis*

Catarrhal inflammation of the accessory sinuses commonly develops during the course of a common cold by spread of infection from the nose. The condition is comparatively trivial and resolution usually occurs, but suppuration may develop due to secondary infection with streptococci, pneumococci or other organisms. If drainage is impaired by oedema of the mucosa around the opening of the sinus, or because of some structural abnormality such as a deviated septum, secondary pyogenic infection is particularly likely to occur and may lead to empyema of the sinus. Pyogenic infection may also spread to the maxillary sinus from the roots of the upper molar or second premolar teeth. When this happens subsequent extraction of the offending tooth may result in the development of an *oro-antral fistula* (an abnormal communication between the antrum and the mouth).

*Symptoms and signs.* The main local symptom of pyogenic inflammation of the nasal sinuses is pain. This is characteristically severe in the middle of the morning and gradually wears off during the day. On examination there is usually an associated area of tenderness. The site of the pain depends on which sinus is involved, as shown in Table II. It is important to notice that *pain due to infection of the maxillary sinus is often described by the patient as toothache.*

TABLE II. Site of pain and tenderness in acute sinusitis

Sinus affected	Site of pain and tenderness
Sphenoidal sinus and posterior ethmoidal air cells	Pain in occipital region or in 'the middle of the head'
Frontal sinus and anterior ethmoidal air cells	Pain in frontal region with tenderness on pressure in supra-orbital region or at inner angle of orbit. Often unilateral
Maxillary sinus	Pain in upper jaw or malar region; often described by the patient as toothache. Usually tenderness on pressure over maxilla or malar bone. Often unilateral

Examination of the nose with a speculum reveals marked swelling and congestion of the mucosa, and pus in the middle meatus of the affected side.

The general symptoms include pyrexia, increased pulse rate and malaise.

In some cases the pent-up muco-pus escapes through the natural opening of the sinus into the nose and the pain is promptly relieved. Sometimes suppuration in the frontal sinus or ethmoidal air cells spreads, giving rise to an orbital, extradural or cerebral abscess, septic meningitis or cavernous sinus thrombosis. These complications are extremely serious.

*Treatment.* The initial treatment consists in the administration of penicillin (or, if this fails, some other appropriate antibiotic), together with conservative procedures, such as steam inhalations and the instillation of nasal drops of 1 per cent ephedrine in normal saline, designed to promote free drainage from the affected sinus. Oily drops, which were once widely used, should be avoided as they interfere with the normal ciliary action of the nasal mucosa. In the later stages saline douches are helpful to deal with hypersecretion of mucus.

If conservative measures fail, operative treatment may be necessary; it should, however, be limited to the simplest procedure which will provide adequate drainage. In the case of the maxillary sinus a small opening is made into the sinus from the inferior meatus (*intranasal antrostomy*). In frontal sinusitis, if there are severe general symptoms or any evidence of meningeal irritation such as neck stiffness, or if there is an abscess pointing externally, the sinus should be opened. Internal drainage of the frontal sinus is now obsolete and if drainage is required it should be provided by making an external opening into the sinus through an incision just below the eyebrow.

### *Chronic sinusitis*

Chronic sinusitis generally results from repeated, or inadequately treated, acute attacks. When the maxillary sinus is infected from a septic tooth, however, the condition may be chronic from the beginning.

*Symptoms and signs.* The symptoms are often not very characteristic. They include headache, intermittent nasal discharge associated with a bad taste in the mouth, and foul breath. In some cases deafness develops.

Diagnosis is difficult or impossible unless certain special methods of examination are employed. These include:

1. *Rhinoscopy*; that is, examination of the nose with a nasal speculum. Preliminary shrinkage of the mucosa with cocaine and adrenalin is often necessary. Pus is looked for in the region of the openings of the various sinuses.

2. *Transillumination.* This method of examination is applicable to the maxillary sinuses only. The patient is taken to a darkened room and a small electric lamp is placed in his mouth. In normal subjects a bright crescentic area is seen in the infra-orbital region and there is a red glow through the pupils. If one maxillary sinus is filled with muco-pus, or if its lining mucosa is greatly thickened, a dark shadow is seen in the infra-orbital region and the red glow through the pupil is diminished.

3. *X-ray examination.* Radiographs should be taken in every case of suspected chronic sinusitis. Increased opacity (Fig. 36), seen as a relatively lighter area in the ordinary X-ray film since this is photographically speaking a 'negative', indicates either fluid or thickened mucous membrane. Fluid with a collection of air above it may cause a definite 'fluid level' (Fig. 37).

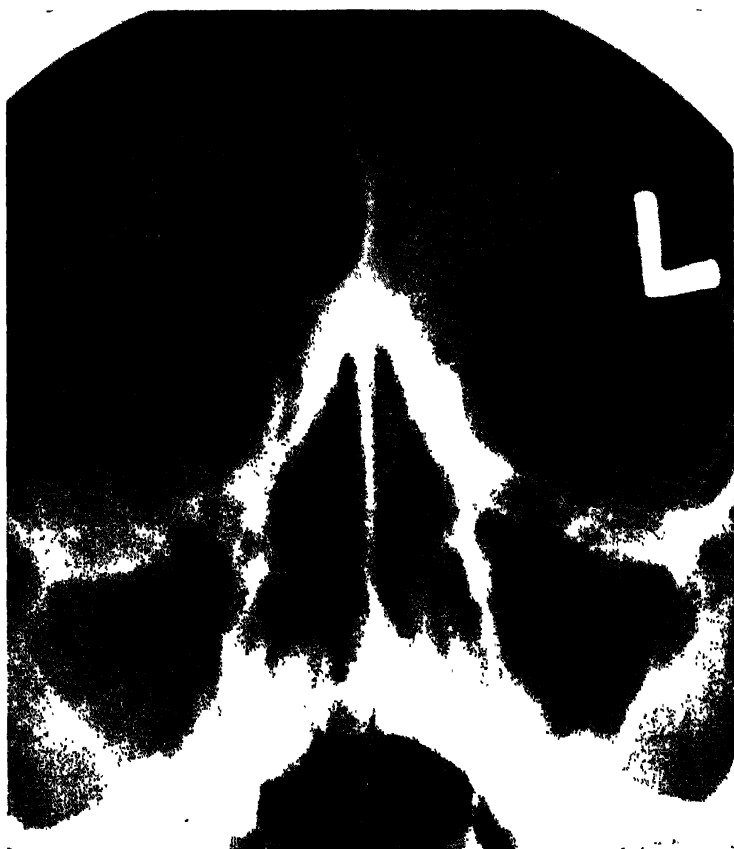


FIG. 36. Radiograph showing opacity of the left frontal sinus.



FIG. 37. Radiograph showing a fluid level in the right antrum.

4. *Diagnostic or proof puncture.* This method of examination is applicable only to the maxillary sinus. Under local anaesthesia a needle is driven from the inferior meatus through the thin bony wall of the sinus and the contents are aspirated.

*Treatment.* The essential treatment is to establish free drainage and aeration of the sinus, and deal with any underlying cause such as a deviated nasal septum or a septic tooth. Chemotherapy, administration of antibiotics and short-wave diathermy are useful auxiliary methods. In acute sinusitis, as we have seen, drainage is commonly established by conservative treatment; in chronic sinusitis, however, operation is often necessary.

Chronic infection of the maxillary sinus may be treated in the first instance by antral lavage. A needle is inserted as for diagnostic puncture and the sinus is washed out by alternately aspirating the contents and injecting normal saline. Lavage is repeated at intervals of about 2 weeks. If infection persists in spite of this treatment operation is required. The upper lip is retracted and the buccal mucosa is incised in the canine fossa to expose the maxilla. An opening is made through the bony wall into the sinus, the diseased mucous membrane lining it is removed, and a counter opening is made into the nose. This is called the Caldwell-Luc operation.

The frontal sinus may be drained, as already described, by an external operation through an incision just below the eyebrow.

The ethmoidal cells and the sphenoidal sinus may be approached either from within the nose or by an external operation. Submucous resection of the nasal septum may have to be undertaken as the first step in the intra-nasal approach to the sphenoidal sinus.

### **Otitis media and mastoiditis**

Inflammation of the middle ear (*otitis media*) is particularly common in childhood, and often occurs during the course of infectious diseases such as measles or scarlet fever. Infection reaches the middle ear from the pharynx, via the Eustachian tube. The organism responsible is usually a streptococcus or pneumococcus.

The inflammation is usually catarrhal at first, but later suppuration may occur.

The main symptoms are pain, deafness and pyrexia. On examination with an aural speculum the tympanic membrane is seen to be hyperaemic; later, as purulent fluid collects in the middle ear, it may appear bulging.

Treatment in the early stages consists in the application of heat and the administration of antibiotics. The condition usually resolves completely but if pain persists or the deafness increases the membrane is incised in a vertical direction at the most prominent part to provide free drainage.

Spread of infection from the middle ear to the mastoid antrum was formerly a common complication, but has become much less frequent since the introduction of penicillin. It is most likely to occur when incision of the membrane is unduly delayed.

The main symptoms of acute mastoiditis are persistence of pyrexia and raised pulse rate despite incision of the tympanic membrane, and recurrence or increase of discharge of pus from the ear. The most important sign is tenderness on pressure over the mastoid process.

The condition usually resolves with appropriate antibiotic therapy, so that operative drainage of the mastoid antrum is nowadays rarely needed. Complications such as meningitis, septic thrombosis in the lateral venous sinus, extradural abscess and cerebral abscess, which were once not uncommon, are now, fortunately, rarely seen.

Chronic mastoiditis is nearly always the result of a previous acute attack. The main symptoms are deafness and persistent discharge from the ear. The condition is dangerous because the infection may spread and give rise to the serious complications mentioned in the preceding paragraph.

If there is a large central perforation of the tympanic membrane the condition may resolve with conservative treatment, but in many cases where there is an attic or posterior marginal perforation some form of mastoid operation has to be undertaken. This involves widely opening the



mastoid antrum and middle ear, and nibbling away the bone between them so that one large cavity is formed from which free drainage can occur. The operation is much more serious than simple drainage for acute mastoiditis and there is danger of injury to the facial nerve. The modern operation of *tympanoplasty* has been designed to conserve the patient's hearing as far as possible. It is frequently performed through an endaural incision, instead of an incision behind the ear as in the old radical mastoid operation, and the ossicles in the middle ear are retained. If, for any reason, the classical complete radical operation is performed, with removal of the drum and all the contents of the middle ear, some permanent impairment of hearing is inevitable. The old adage that prevention is better than cure thus applies very strongly to chronic mastoid suppuration.

### **Tonsillitis**

#### *Surgical anatomy*

The tonsils—properly known as the faucial or palatine tonsils—form part of a widely dispersed mass of lymphoid tissue which surrounds the entrance to the pharynx. They are situated between the anterior and posterior pillars of the fauces and are easily visible if the tongue is depressed with a spatula.

The lymphoid tissue of the tonsil is enclosed in a capsule of connective tissue, and this in turn is covered on its superficial aspect by stratified squamous epithelium. The surface is not smooth, but shows numerous shallow epithelial-lined pockets known as the tonsillar crypts.

#### *Acute tonsillitis*

Simple catarrhal tonsillitis commonly occurs in association with catarrhal pharyngitis.

The severer types of tonsillitis are spoken of as *lacunar* (sometimes called follicular) and *parenchymatous* tonsillitis, but these terms have no great merit. In the lacunar type the crypts become filled with pus and epithelial debris; in the parenchymatous form inflammation involves the whole tonsil. The causal organism is usually the streptococcus but pneumococci and staphylococci may also be found.

Acute tonsillitis is particularly common in children and young adults; it often occurs in the early stages of measles and other infectious diseases.

*Symptoms and signs.* The main symptoms are pain in the throat and pyrexia. The pain often radiates to the ears and is made worse by swallowing. In some cases the patient has a rigor.

Acute tonsillitis must be distinguished from diphtheria, Vincent's angina (Chapter XII) and glandular fever, and from ulceration associated with severe blood diseases such as acute leukaemia and agranulocytosis. Confusion with the mucosal lesions of secondary syphilis (Chapter XIII) should not occur since these have a characteristic appearance and are relatively painless.

*Treatment.* Aspirin is given to relieve the pain, and penicillin to combat the infection. Resolution usually occurs rapidly, but occasionally a peritonsillar abscess (page 114) develops. If the condition is recurrent tonsillectomy should be performed some weeks after the acute inflammation has subsided.

### *Chronic tonsillitis*

Chronic tonsillitis is usually the result of repeated acute attacks. Mere enlargement of the tonsils is no evidence of chronic infection; indeed in many cases infected tonsils are quite small. The characteristic symptoms and signs of chronic tonsillitis are: (a) a history of recurring acute attacks; (b) the presence of pus and epithelial debris in the tonsillar crypts, and (c) enlargement of the cervical lymph nodes, especially the jugulo-digastric nodes which are situated behind the angle of the jaw on each side.

Chronically inflamed tonsils are commonly found in children with tuberculous cervical lymphadenitis (Chapter XIII).

*Treatment.* Tonsillectomy should be performed if the diagnosis is definite. In young children the operation is sometimes done with a guillotine, but dissection is preferable, and is always necessary in older patients. Severe haemorrhage is an occasional post-operative complication.

Indiscriminate removal of tonsils, like indiscriminate extraction of teeth, has sometimes been practised in an attempt to eradicate septic foci (Chapter XI), but these operations should never be performed unless there is definite evidence of disease in the structures concerned.

## **Septicaemia and Pyaemia**

It is not uncommon for bacteria to enter the circulation from a local focus, and when this happens infection may be widely disseminated. Sometimes the organisms enter the blood stream intermittently and are quickly eliminated, and the condition is then termed *bacteraemia*. Alternatively, organisms may continue to be demonstrable in blood cultures for some time and the condition is then termed *septicaemia*. It used to be thought that septicaemia implied that the organisms were proliferating in the

blood stream but it is now believed that it may result simply from the continuous entry of new organisms from the local focus.

Septicaemia may occur with a wide variety of organisms including streptococci, staphylococci, pneumococci, gonococci, *E. coli*, *S. typhosa*, *Cl. Welchii* (the main organism responsible for gas gangrene), *P. pestis* (the organism responsible for pneumonic and bubonic plague) and *B. anthracis*.

Clinically the onset is usually rapid and may be heralded by a rigor. The patient is acutely ill, with a high temperature (for example, 105° F.), rapid pulse, dry furred tongue, restlessness and sometimes delirium. The skin is hot and dry, and may show a rash; the urine is scanty and often contains albumen, and there may be diarrhoea. There is usually a considerable leucocytosis, but sometimes, in the later stages or in previously debilitated patients, the bone marrow is unable to keep up the supply of leucocytes, and leucopenia results. The diagnosis is confirmed by obtaining a positive blood culture.

#### *Treatment of septicaemia*

The local focus must receive appropriate treatment, including drainage of collections of pus under tension, and occasionally, in extreme cases, amputation of a limb.

General measures include the provision of an adequate fluid intake by mouth, by rectum or intravenously, and specific treatment with an appropriate antibiotic (Chapter XI). Fortunately the organisms concerned are often sensitive to one or more of these substances; if they are completely insensitive the chances of recovery are small.

### **Pyæmia**

Pyæmia is a condition in which particles of infected material (termed *septic emboli*) enter the circulation and are carried to distant parts of the body where they give rise to multiple abscesses. These emboli consist of clumps of pyogenic organisms, detached pieces of infected blood clot from a thrombus in a vein, or detached vegetations from the heart valves in patients with infective endocarditis.

If the primary focus of infection is connected with the systemic venous system the abscesses occur mainly in the lungs; if with the portal venous system the abscesses occur mainly in the liver. If the primary focus is in a heart valve or in the lungs secondary abscesses may occur almost anywhere in the body, but are most common in the kidneys and in joints.

Initially the clinical features resemble those of septicaemia, except that the temperature swings more and rigors recur frequently. After about a week, if the patient survives, the secondary abscesses appear.

Treatment follows the same lines as in septicaemia. In addition, surgical ligation of the vein which is the source of the emboli may be undertaken in some cases; in pyaemia due to septic thrombosis of the transverse sinus, for instance, the internal jugular vein may be ligated. In the later stages secondary abscesses may require aspiration or open drainage.

### **Focal Sepsis**

Many disorders have, at one time or another, been attributed to a focus of infection somewhere in the body. Such claims have sometimes been put forward without any evidence to support them, and in consequence some surgeons have tended to discount the importance of focal sepsis as a cause of disease elsewhere; this reaction has however gone too far.

There is in the first place no doubt that local foci may give rise to bacteraemia and hence to secondary foci in other sites. Endocarditis, for example, may develop in this way.

In addition, there are various disorders, including rheumatic fever, rheumatoid arthritis, glomerulonephritis and some forms of fibrositis, which are not due directly to the dissemination of bacteria, but in which focal infection, usually if not always streptococcal, in the pharynx, in relation to the teeth (gingivitis and apical osteitis), or less commonly in other sites, appears to play an etiological role. The mechanisms involved have not been fully elucidated but in some cases at least the disease appears to result from an immunological reaction against a complex antigen formed by the combination of material produced by the infecting organisms with some component of the patient's tissues.

Focal sepsis has sometimes been diagnosed without any justification, and a considerable number of normal teeth and tonsils have unfortunately been removed in misguided attempts to cure a patient's lumbago or 'rheumatism'. There is no excuse for this. On the other hand it is important to treat genuine focal sepsis energetically. In particular, acute streptococcal pharyngitis should be treated with adequate doses of penicillin, and chronically infected tonsils should be removed under penicillin cover. Root-filled teeth should be regarded with suspicion and extracted if radiological examination reveals associated apical infection.

## *Chapter XI*

# CHEMOTHERAPY AND ANTIBIOTICS

## IN THE TREATMENT OF INFECTION

*Chemotherapy* means treatment with complex organic drugs of known molecular structure. Many chemotherapeutic agents have been synthesized from quite simple substances; others have been derived from living organisms of various kinds, though the chemical structure of the natural substance may have been modified in the laboratory. Chemotherapy began with Ehrlich's search for a cure for syphilis, which culminated in the discovery of *Salvarsan* (1910) and *Neosalvarsan*. Developments followed rapidly in the field of tropical medicine, but the most dramatic advance came in (1910) when Domagk introduced *prontosil rubrum*, the first of the sulphonamide group of drugs.

*Antibiotics*, strictly speaking, are substances produced by living organisms which are toxic for other living organisms; the term antibiotic is often used loosely, however, to cover all antibacterial chemotherapeutic agents.

Penicillin, which was the first antibiotic to be used therapeutically, was discovered by Fleming in 1910 when he observed that the growth of a culture of staphylococci was inhibited by the mould *penicillium notatum*. This observation aroused little interest, however, until Florey and his associates, as a result of extensive investigations, beginning in 1910, succeeded in concentrating penicillin and preparing stable salts which could be used therapeutically. A few other antibiotics, notably streptomycin and neomycin, which we owe to Waksman, were discovered by independent research workers; the rest were developed by the pharmaceutical industry, mainly as the result of screening a vast number of fungi and bacteria for antibiotic activity. In this way many hundreds of antibiotics have been found, but only a small proportion of these have turned out to be therapeutically useful.

### **The Sulphonamides**

Following Domagk's work it was soon shown that the antibacterial action of prontosil was due not to this substance itself but to para-aminobenzenesulphonamide (sulphanilimide), which is formed from it in the body. Sulphanilimide itself was found to be effective only against haemolytic streptococci, but by substituting different chemical groupings in the

molecule a number of compounds, known collectively as the sulphonamides, having a somewhat wider range of anti-bacterial activity, were produced.

The sulphonamides are *bacteriostatic* rather than *bactericidal*; that is, their main effect is to prevent the multiplication of bacteria rather than to kill them. The action of the drugs is antagonized by substances containing the p-amino-benzoic group (e.g. novocaine) and by unidentified substances in pus and necrotic tissue.

### **Indications and dosage**

The sulphonamides which are currently used fall into two main groups:

1. Sulphonamides which are readily absorbed and distributed by the blood stream, and can therefore be used to treat infection in various parts of the body. *Sulphadimidine* is the safest and most generally useful member of this group. In recent years several long-acting sulphonamides have been introduced, mainly for use in the treatment of infections of the urinary tract, but they are as yet unproved.

2. Sulphonamides such as *succinyl-sulphathiazole* and *phthalyl-sulphathiazole*, which when given by mouth are absorbed to only a slight extent, and whose site of action is therefore virtually restricted to the alimentary tract.

With the development of antibiotics the indications for using sulphonamides have narrowed considerably.

Sulphadimidine is often effective in the treatment of infections due to haemolytic streptococci, and may also be of value in the treatment of infections due to gram negative bacilli including *E. coli*, *B. Proteus*, and *Ps. pyocyaneus*, especially in the urinary tract.

Sulphadimidine is normally given orally, but an initial intravenous injection of the sodium compound is occasionally used. An average course of treatment for an adult would be 2 to 3 grammes initially, followed by 1 to 1.5 grammes every 4 to 6 hours for a few days.

The indiscriminate use of sulphonamides is futile and may be dangerous. Clinical evidence of the nature of the infection may be sufficient to justify starting treatment, but before the first dose of the drug is given steps should be taken to obtain material suitable for bacterial culture so that the sensitivity of the organisms both to sulphonamides and to a variety of antibiotics may be determined. Occasionally, when using sulphonamides of the first group, failure occurs even when the organism has been proved to be sensitive; this usually means that the amount of the drug absorbed is insufficient to raise the concentration in the blood to an adequate level.

In children and infants the dose is calculated from the fact that in proportion to their weight children tolerate twice as much sulphonamide as adults. Thus where one would give an initial dose of 2 grammes to a 10 stone adult one should give  $2 \times \frac{1}{10} \times 2 \approx 1\frac{1}{2}$  grammes to a child weighing 4 stone.

Sulphonamides of the first group were at one time used locally for the treatment of infected wounds and burns, but this practice is dangerous (*v. infra*) and has now been abandoned.

Sulphonamides of the second group are used to reduce the intestinal flora prior to operations on the colon, and also in the treatment of bacillary dysentery and diverticulitis.

### **Dangers of sulphonamide therapy and precautions to be observed**

Sulphonamide therapy may have a number of unpleasant or dangerous consequences, but these can be largely avoided by taking appropriate precautions.

Before treatment is begun the patient should be asked whether he has had sulphonamides recently, and whether he has at any time shown evidence of unusual sensitivity to any of these drugs. If there is a history of sensitivity a small test does (0.1 gramme) should be given, and if toxic manifestations occur some other form of treatment must be used.

In addition, as described below, special precautions must be taken to avoid particular complications. These complications may be grouped under five headings, as follows:

1. *Minor symptoms.* These include general malaise, headache, nausea and vomiting.

2. *Urinary complications.* Crystals of the drug itself, or of an acetyl compound formed from it in the body, may be precipitated in the kidneys, ureters or bladder; this is most likely to occur if the urine is acid in reaction. The crystals may block the renal tubules or the ureters.

Symptoms may appear at any time during the course of treatment. They include pain in the loins sometimes radiating to the groins, haematuria and diminished output of urine. In severe cases there may be complete anuria.

This complication was fairly common when sulphamerazine, sulphadiazine and sulphathiazole were used, but rarely occur with sulphadimidine. It is, however, wise to ensure that the patient has an adequate fluid intake and, if his urine is acid, sodium bicarbonate and sodium citrate (1 gramme of each with each dose of sulphonamide) should be given. If

symptoms occur the treatment is to stop the drug, apply heat to the loins, and administer alkalis and fluids. If anuria develops an attempt is made to relieve the obstruction by passing catheters up the ureters; fluids are not pushed until this has been done.

Very occasionally sulphonamides cause toxic damage to the epithelial cells of the tubules of the kidney and consequent renal failure. In severe cases the patient may require treatment with the artificial kidney.

3. *Blood disorders.* These include various types of anaemia, purpura, leucopenia and agranulocytosis.

Agranulocytosis is especially important. It is a serious and often fatal condition in which the formation of polymorphonuclear leucocytes (sometimes called 'granulocytes') is greatly impaired. The symptoms include fever and severe sore throat; examination of the blood shows a total white count of less than 2,500 cells per cu. mm. and a neutrophil count of less than 1,000 cells per cu. mm. Sometimes the total white count falls as low as 100 cells per cu. mm.

Agranulocytosis rarely occurs unless sulphonamide therapy has been continued for 10 days or longer, and is usually preceded by leucopenia without other symptoms. It is therefore a wise precaution to limit the period of administration of sulphonamide drugs to about a week or, if treatment is continued for longer than this, to perform daily leucocyte counts. If leucopenia develops the drug should be stopped at once. In true agranulocytosis, in addition to stopping sulphonamides, pentose nucleotide is given to stimulate the bone marrow and an appropriate antibiotic to combat the throat infection. Pyridoxin is also said to be of value. In severe cases repeated blood transfusion may be tried, but the results are often disappointing.

4. *Skin eruptions.* These are much less frequent now that the local application of sulphonamides has been abandoned, but occur occasionally after oral administration, especially when sulphonamides have been given on a previous occasion. The rash usually appears after 7 to 9 days, and is most often a discrete, papular, erythematous eruption; it may be accompanied by fever, oedema, albuminuria or jaundice. Occasionally severe exfoliative dermatitis occurs.

If sulphonamides are used locally in the treatment of skin diseases a weeping eczematous rash may develop.

As usual, the first and most essential step in treatment is to stop the administration of the drug.

5. *Drug fever.* Fever resulting from the administration of sulphonamides is commonly, though not always, associated with skin rashes and other evidence of 'hypersensitivity'.



Fever due to the drug must be distinguished from fever due to organisms which the drug has failed to control.

6. *Foetal abnormalities.* There is reason to fear that some of the long-acting sulphonamides, in particular sulphadimethoxypyrimidine, sulphadimethoxine and sulphamethoxydiazine, are liable to cause foetal abnormalities. It is arguable whether these drugs should ever be used; certainly they should not be given during pregnancy.

### **Antibiotics. General Considerations**

#### **Structure and mode of action**

From the point of view of chemical structure the antibiotics in use today appear at first sight to be a very heterogeneous collection of substances, but closer examination suggests that they represent variations on a small number of biochemical themes. A curious but important fact is that antibiotics with very different chemical structures may have a very similar range of antimicrobial activity.

Some antibiotics, including the tetracyclines and chloramphenicol, like the sulphonamides are bacteriostatic rather than bactericidal; that is, their main effect is to prevent multiplication of bacteria rather than to destroy them. Others, however, notably penicillin, streptomycin and vancomycin, are bactericidal.

Most antibiotics today are produced commercially by large-scale culture of the appropriate organism, followed by chemical processes designed to isolate the active substance and in some cases to modify the structure of the molecule in a particular way. Chloramphenicol, however, though originally obtained from *Streptomyces venezuelae*, can now be synthesized in the laboratory.

The discovery of new antibiotics by empirical screening procedures has advanced much more rapidly than our understanding of their mode of action. It is now known, however, that penicillin interferes with the synthesis of material which makes up the cell wall of sensitive organisms, while chloramphenicol interferes with protein synthesis within the bacterial cell.

#### **Resistance to antibiotics**

Some species of bacteria may be completely resistant to a particular antibiotic, while other species may vary greatly in their resistance, some strains being highly sensitive and others resistant. Unfortunately, the range of usefulness of many antibiotics has been found to decrease as they come to be more widely used because the frequency with which resistant strains

are encountered increases. Today, for example, the proportion of patients with staphylococcal infections who can be successfully treated with ordinary penicillin is very much smaller than it was when penicillin was first introduced.

There are three main ways in which an organism may resist the inhibitory effect of an antibiotic. In the first place, particular metabolic pathways which are interrupted in sensitive organisms by the antibiotic may be absent in resistant strains. Secondly, the antibiotic may be unable to penetrate the cell wall of the organism. Thirdly, the organism may produce an enzyme which destroys the antibiotic. A well-known example of the last of these mechanisms is the production by some strains of staphylococci of an enzyme known as *penicillinase* which destroys penicillin.

Individual organisms which are initially sensitive to a particular antibiotic may become resistant by the process known as *enzymic induction*, or as the result of changes in their genetic constitution due to *mutation* or *gene recombination*. Enzyme induction implies a specific increase in the rate of formation of an enzyme which is responsible in some way for making the antibiotic ineffective; a familiar example is provided by the observation that staphylococci which have produced little or no penicillinase may begin to produce large amounts of this enzyme after coming in contact with penicillin. The induced character is not genetically transmitted but, on the other hand, it is not immediately lost if the antibiotic is withdrawn because some of the enzyme persists for quite a time even though no more is formed. Genetic changes are, of course, inherited. They occur infrequently and in the absence of antibiotic the number of resistant organisms remains small. In the presence of an antibiotic to which the unchanged organisms are sensitive, however, the resistant organisms have a selective advantage and are likely to become predominant.

### **Dangers of antibiotic therapy**

The dangers of antibiotic therapy may be considered from the point of view of the patient, the doctor or dentist, and the community.

So far as the patient is concerned there are three main dangers: (a) the emergence of resistant strains of organisms; (b) secondary colonization of the lesion by organisms of various species which may be hard to eradicate, and (c) toxic effects of the antibiotic. The types of toxic effect which may occur with different antibiotics will be considered later.

The main danger for the doctor or dentist is that he may be lulled into a false sense of security, and in consequence may become careless and fail to take proper aseptic and antiseptic precautions.

The danger to the community is that the extensive use of a particular antibiotic may lead to resistant strains of organisms becoming so predominant that the antibiotic in question ceases to be of much value.

### **The choice and clinical use of antibiotics**

Antibiotics have been of benefit to millions of people but to use them unnecessarily and indiscriminately is both wasteful and dangerous. Whenever possible the organisms responsible for the patient's infection should be identified, and their sensitivity to a range of antibiotics should be determined. Sometimes, if the patient is seriously ill it may be justifiable to start treatment before this has been done, basing the choice of antibiotic on clinical evidence of the nature of the infection; in this event, however, every attempt should be made to obtain material suitable for bacterial culture before the first dose is given.

The prophylactic use of antibiotics is often ill-advised but is justified under certain circumstances. Thus, for example, penicillin may be given to minimize the risk of osteomyelitis of the jaw and generalized infection when septic teeth require to be extracted.

### **Penicillin and Related Antibiotics**

Several hundreds of members of the penicillin family have been produced, but only a few are currently used therapeutically, and these may be subdivided into four main groups as shown in Table III.

#### **Benzyl penicillin**

Benzyl penicillin, otherwise known as penicillin G, was the first penicillin to become available for therapeutic use, and is still of great importance. *Procaine-penicillin* is a compound of procaine and benzyl penicillin, which causes less pain on intramuscular injection than ordinary benzyl penicillin and is more slowly absorbed.

The dosage of benzyl penicillin is expressed in units (known as Oxford units) which are defined in terms of activity *in vitro* against a particular strain of staphylococci. Pure crystalline penicillin has a potency of 1,650 units per milligramme, but the potency of present-day commercial penicillin is only about 1,000 units per milligramme.

Benzyl penicillin is rapidly destroyed by heat, and preparations should if possible be stored in a refrigerator. It is also destroyed by acids, alkalis and many antiseptics.

TABLE III. Penicillin and related antibiotics

Group	Approved name	Pro-prietary names	Main indications	*Route of administration	†Dosage (adults)
1. Original form of penicillin	Benzylpenicillin (penicillin G) Procaine benzylpenicillin	Too numerous to mention	See text	I.M.	See text
2. Penicillins for oral administration having a similar range of anti-bacterial activity to benzylpenicillin	Phenoxy-methylpenicillin (penicillin V) Phenethicillin Propicillin Phenbenicillin	Too numerous to mention Broxil Brocillin Penspek	Alternative to benzyl-penicillin when injection is contra-indicated or inconvenient	Oral	125-250 mg. every 4-6 hours
3. Penicillins resistant to staphylococcal penicillinase	Methicillin Cloxacillin	Celbenin Orbenin	Staphylococcal infections resistant to benzylpenicillin	I.M. Oral or I.M.	1,000 mg. every 6 hours 500 mg. every 6 hours (oral) 250 mg. every 6 hours (I.M.)
4. Broad spectrum penicillin & cephalosporins	Ampicillin Cephaloridine	Penbritin Ceporin	Infection due to various gram-ve bacilli (excluding <i>Ps. pyocyaneus</i> . Effective also against organisms sensitive to benzylpenicillins Many gram-positive & gram-negative organisms including penicillinase-producing staphylococci & coliforms	I.M.	250 mg. every 6 hours 250-500 mg. every 12 hours

mg. = milligrams.

\*I.M. = intramuscular injection.

†The doses stated are for adults. Smaller doses should be used in children.

*Indications*

It is impossible to be certain whether a given infection will respond to benzyl penicillin without isolating the causal organism and performing a sensitivity test. Broadly speaking, however, benzyl penicillin is effective against the following organisms:

1. Many pathogenic cocci, both gram-positive and gram-negative, including *streptococcus pyogenes*, *streptococcus pneumoniae*, *Neisseria gonorrhoea*, and strains of *Staphylococcus pyogenes* which do not produce penicillinase.
2. Most gram-positive bacilli, including *Clostridium welchii* and *Bacillus anthracis*, but excluding *Mycobacterium tuberculosis*.
3. The organism causing actinomycosis (most strains).
4. The organisms causing Vincent's angina.
5. The *Spirochaeta pallida* of syphilis.

*Administration and dosage*

Benzyl penicillin is normally given by intramuscular injection. The dosage varies somewhat according to the condition.

*Generalized infections.* In treating a generalized infection such as staphylococcal or streptococcal septicaemia it is necessary to maintain the concentration of penicillin in the blood at a high level. Since penicillin is rapidly absorbed and excreted this means that injections must be given every 3 or 4 hours. Typically, an initial injection of 1,000,000 units is followed by injections of 300,000 units every 3 hours. Treatment is continued for some days after the patient has become afebrile.

*Localized infections.* In treating a localized infection such as a carbuncle or Vincent's Angina three methods are available:

(1) The plan described for generalized infections may be followed but with a smaller initial dose; for example, 300,000 units every 4 hours. Treatment is continued until the inflammation subsides; this usually takes a few days.

(2) Procaine penicillin may be used. As this is slowly absorbed an injection of 300,000 units once or twice every 24 hours may suffice to maintain an effective blood concentration. 300,000 units of ordinary benzyl penicillin may be given with the first injection in order to achieve an effective blood concentration quickly.

(3) Large doses (1,000,000 units) of ordinary benzyl penicillin may be given once or twice every 24 hours. This does not maintain a constant high blood concentration but this does not necessarily matter greatly so far as

localized lesions are concerned provided that a high concentration is maintained in the tissues.

*Prophylactic use of penicillin.* It is good practice to give 300,000 units of ordinary penicillin and 300,000 units of procaine penicillin half an hour before extracting impacted molars or teeth with associated apical infection. This helps to minimize the risk of subsequent osteomyelitis of the jaw. The injection of procaine penicillin may be repeated after 24 hours. Penicillin is also used prophylactically in patients subject to recurrent streptococcal sore throat to reduce the risk of their developing rheumatic fever or nephritis, and sometimes also in patients with these diseases to reduce the risk of exacerbations.

*Other uses.* After intramuscular injection, penicillin is absorbed into the blood stream and carried to the tissues. It does not, however, pass from the blood to the cerebrospinal fluid, the cavities of joints or the aqueous humour of the eye. For this reason special methods of administration are sometimes used; in septic arthritis, for instance, penicillin may be injected directly to the affected joint.

Topical application of penicillin powder or creams is still sometimes used in the treatment of burns, but the risk of sensitization is considerable and this practice should be abandoned.

The use of benzyl penicillin lozenges or chewing wafers for treating local lesions in the mouth should also be regarded as obsolete because, as the penicillin-sensitive organisms are destroyed, the number of insensitive organisms increases and the patient may develop a troublesome stomatitis.

#### *Dangers. Precautions to be observed*

The main error in using penicillin is to rely on the drug alone when surgical drainage of pus is required; the main danger is that as sensitive organisms are destroyed penicillin-resistant strains may gain a foothold.

Toxic effects, though not very common, occur more often than was once thought. They include symptoms resembling serum sickness, skin eruptions which may be erythematous, urticarial or occasionally exfoliative, and fever. Pain at the site of injection is common except when procaine-penicillin is used, but can hardly be called a toxic manifestation.

If a skin eruption does appear penicillin should be stopped and some other antibiotic substituted. The patient's discomfort can sometimes be relieved by giving one of the antihistamine drugs such as *anthisan*.

Strict asepsis must be observed when giving penicillin injections. Many cases have occurred in which resistant organisms have been introduced as a result of lack of care in this respect.

**Oral penicillins having a similar range of antibacterial activity to benzyl penicillin**

The members of this group are resistant to acid and in consequence are not destroyed in the stomach if given by mouth. The first one to be used was *phenoxymethylpenicillin*, otherwise known as penicillin V. This is similar to (though not absolutely identical with) benzyl-penicillin so far as range of antibacterial activity is concerned, but offers the convenience of oral administration. The other members of the group (Table II) were produced later by replacing the methyl group in the molecule of phenoxymethylpenicillin by an ethyl, a propyl or a benzyl group. They have generally similar properties to phenoxymethylpenicillin. *Phenethicillin* and *propicillin* are sometimes effective against staphylococci which produce small amounts of penicillinase, but are very inferior in this respect to the members of the next group (methicillin and cloxacillin).

The usual dose for phenoxymethylpenicillin and other members of the group is 125 to 250 mg. every 4 to 6 hours.

**Penicillins resistant to staphylococcal penicillinase**

The development of penicillins resistant to staphylococcal penicillinase was a major advance which has greatly increased our capacity to deal with infections due to staphylococci resistant to benzylpenicillin.<sup>1</sup> It is to be hoped that this great achievement will not be nullified by irresponsible use of these drugs leading to the emergence and eventual predominance of strains of staphylococci which are resistant to them. The first member of this group to be developed was *methicillin*, which is given by intramuscular injection in a dosage of 1 gramme every 6 hours. *Cloxacillin*, which was developed more recently, may be given either by mouth (500 mg. every 6 hours) or by intramuscular injection (250 mg. every 6 hours). These drugs should be used only for the treatment of severe infections in which staphylococci resistant to benzylpenicillin are playing a major role.

**Broad spectrum penicillins**

*Ampicillin*, which came into use in 1910, differs from previous penicillins in being effective against a wide range of gram-negative bacilli, though it is also effective against organisms sensitive to benzylpenicillin. It is not effective against penicillinase-producing staphylococci or penicillinase-producing coliforms, or against *Pseudomonas pyocyaneus*. It is given by intramuscular injection in a dosage of 250 mg. every 4 hours.

<sup>1</sup> Such organisms are commonly referred to briefly as 'penicillin-resistant staphylococci'.

*Cephaloridine*, which is a modification of Cephalosporin C, and which should perhaps be regarded as a relative of the penicillins rather than a member of the family, is active against a wide range of gram-positive and gram-negative organisms, including penicillinase-producing staphylococci and coliforms.

### Streptomycin

Streptomycin, obtained from the fungus *streptomyces griseus*, was introduced for the treatment of tuberculosis in 1910. For this purpose it is always used in combination with other agents, in particular p-aminosalicylic acid and isoniazid, because in this way the danger of drug-resistant strains of tubercle bacillus emerging is very small, whereas if streptomycin is used alone this is virtually certain to happen.

Streptomycin is also fairly active against *E. Coli* and various other gram-negative bacilli, though nowadays infections of this nature are usually treated with ampicillin or one of the tetracyclines.

Streptomycin is normally given by intramuscular injection, but intrathecal injections are used in tuberculous meningitis. The main toxic manifestations are tinnitus, nerve deafness and vertigo, but gastro-intestinal disturbances, skin rashes, albuminuria and bone marrow depression may also occur. The likelihood of these manifestations developing appears to depend on the total quantity of streptomycin administered; 1 gramme twice daily may be given for 4 or 5 days, but if prolonged treatment is needed smaller doses must be used.

### The Tetracyclines

There are two members of this family in the 1910 British National Formulary: *tetracycline* and *oxytetracycline* (Terramycin). *Chlortetracycline* (Aureomycin), which was widely used at one time is now obsolete, but a chemically related compound *demethylchlortetracycline* (Ledermycin) has recently been introduced.

Tetracycline and oxytetracycline are effective against a wide range of gram-positive and gram-negative bacteria. They have been of great importance, especially for the treatment of infections due to penicillin-resistant staphylococci and gram-negative intestinal bacilli, but are gradually being superseded for these purposes by methicillin and ampicillin respectively.

The tetracyclines have the advantage of being effective by mouth, but preparations for intramuscular and intravenous injection are available, and are used for example in treating patients with peritonitis. The usual



oral dose of tetracycline and oxytetracycline is 250 to 500 mg. every 6 hours for not more than 7 days. The recommended dose of demethylchlor-tetracycline is 150 mg. every 6 hours.

Nausea, vomiting and diarrhoea are fairly common toxic effects but are seldom dangerous and can be minimized by giving milk or sodium bicarbonate with capsules. Stomatitis and glossitis are also fairly common complications. They used to be attributed to deficiency of vitamins of the B complex resulting from the destruction of intestinal bacteria which normally synthesize these substances, but are more often due to monilial infection.

The most dangerous complication of tetracycline therapy is staphylococcal enteritis. In this condition staphylococci gain entrance to the gut and proliferate rapidly in the absence of competition from the normal bacterial inhabitants. This process, which is termed superinfection, may also occur with organisms other than staphylococci.

### **Chloramphenicol**

Chloramphenicol (sometimes called chloromycetin), which was obtained from the fungus *streptomyces venezuelae* in 1910 and has since been synthesized, is effective against many gram-negative and some gram-positive bacteria. In view of the discovery that a small proportion of patients treated with chloramphenicol develop a fatal form of anaemia, however, the drug should only be used if the infection is a serious one and the organisms are insensitive to other antibiotics. This means in practice that chloramphenicol is rarely indicated in peritonitis, infections of the urinary tract and chronic bronchitis for which it was formerly prescribed, though it is still used in the treatment of typhoid fever. As far as possible the dose of chloramphenicol should not exceed 2 grammes daily and the drug should not be given for more than 5 days.

### **Erythromycin, Novobiocin, Fucidin and Vancomycin**

These antibiotics are grouped together because they have been useful mainly for treating infections due to penicillin-resistant staphylococci.

Erythromycin, novobiocin and fucidin are given orally every 6-8 hours, the total daily dose (for adults) being 1-2 grammes. Novobiocin is a moderately toxic drug which causes severe skin rashes in some patients and is rarely indicated nowadays; the other two rarely cause serious toxic manifestations but resistance to erythromycin may develop very quickly.

Vancomycin is not absorbed from the alimentary tract and is normally given by slow intravenous drip, the total daily dose (for adults) being 1-2

grammes; it may however be given orally for the treatment of acute staphylococcal enteritis. Vancomycin is liable to cause severe toxic manifestations, including phlebitis, skin rashes, renal damage and nerve deafness. It is excreted normally in the urine and is particularly dangerous in patients with poor renal function unless special precautions are observed. It should be reserved for treating severe staphylococcal infections (especially staphylococcal septicaemia) resistant to other antibiotics including methicillin.

### Other Antibiotics

A mixture of *neomycin*, *polymyxin* and *bacitracin* in the form of powder is used topically by some surgeons, for example in operation wounds or for the local treatment of burns. Others condemn this practice, but in the writer's experience it has proved safe and sometimes beneficial. *Neomycin* is also given by mouth as an alternative to phthalylsulphathiazole, or in combination with it, preparatory to operations on the colon, but *polymyxin* and *bacitracin* are so nephrotoxic that they should rarely be used except topically.

*Kanamycin* resembles neomycin but is somewhat less toxic and is used parenterally (intramuscularly) in treating infections due to gram-negative bacilli resistant to other antibiotics; it is however liable to cause nerve deafness and should be used only when indicated and then with due care.

*Cycloserine* is used in the treatment of tuberculous infections resistant to the standard combination of streptomycin, p-aminosalicylic acid and isoniazid.

*Colistin* (Colomycin), which is closely related to polymyxin, is at present the best agent for treating infections due to *Pseudomonas pyocyaneus*, though it is not always very effective.

*Griseofulvin* is given orally in the treatment of fungus infections of the skin.

*Nystatin* is used locally in the treatment of monilial infections of the mouth and pharynx.

## *Chapter XII*

### SPECIFIC INFECTIOUS DISEASES—I

In this and the following chapter we shall discuss some important specific infectious diseases.

#### **Vincent's Angina**

Vincent's angina is caused by the *B. fusiformis* and a *spirochaete* acting together. There is often a superadded streptococcal infection though this rarely appears during the first day or two. The bacillus and the spirochaete are both gram-negative, but are best stained with carbol fuchsin or some other simple stain.

The onset is often acute, with pain which the patient may describe as toothache. On examination in the early stages there is marked gingivitis with areas of ulceration around the teeth. Later the process spreads, and there may be widespread ulceration in the mouth and pharynx, especially in the region of the tonsil. The ulcers are shallow, with irregular but fairly sharp edges, and a yellow membranous slough in the floor. This slough is adherent and attempts to remove it may cause bleeding. The breath has a characteristic foetor. If there is associated streptococcal infection cervical lymphadenitis or even cellulitis may follow.

#### **Diagnosis**

The diagnosis is suggested by the clinical findings and confirmed by demonstrating the specific organisms in a smear.

When there is ulceration of the tonsil Vincent's angina must be differentiated from acute tonsillitis (Chapter X) and diphtheria (*vide infra*). Ulceration due to secondary syphilis (Chapter XIII) should be easily distinguished on account of its characteristic appearance and the absence of pain. In acute leukaemia it is not uncommon to find an ulcerative gingivitis which gives a positive Vincent's smear and the possibility of this association must be borne in mind.

#### **Treatment**

In acute cases the patient is given penicillin by intramuscular injection and hydrogen peroxide mouth washes. For reasons stated in the last chapter

penicillin lozenges should not be used. When the acute phase has subsided scaling of the teeth is undertaken if indicated.

In chronic cases local application of 10 per cent chromic acid to the ulcers is useful. Before this is done the mouth should be syringed with a pint of warm lather made with one of the oxidizing soap powders, or with hydrogen peroxide; this serves to remove accumulations of mucus. In addition to local treatment penicillin may be given by intramuscular injection.

### Diphtheria

Diphtheria is due to infection with the *Klebs-Löffler* bacillus (*Corynebacterium Diphtheriae*). This is a gram-positive organism having a characteristic beaded appearance best shown by Neisser's stain.

The disease is transmitted directly or indirectly from patients or healthy carriers. Infection is most commonly conveyed by droplets of moisture ejected by coughing, sneezing or talking; indirect infection is less common and is due to contaminated drinking utensils, throat spatulas, etc. There is an incubation period of 2 to 7 days. Babies appear to be immune, but children from 2 years onwards are usually highly susceptible unless active immunization has been carried out as described below.

Diphtheritic infection usually involves the mucous membrane of the fauces, larynx or nose; occasionally, however, it occurs on the skin, external genitals or conjunctiva, or in wounds. On mucous surfaces, and sometimes also when the skin is involved, there is exudation of fluid rich in fibrin and necrosis of the superficial layers of the epithelium. The exudate and necrotic material together form a tough, white adherent structure known as a diphtheritic or *false* membrane. Removal of this membrane usually causes bleeding.

Whatever the site of the local lesion serious general symptoms and signs may develop due to absorption of the exotoxin formed by the organisms. These include circulatory failure, albuminuria and muscular paralysis.

*Faucial diphtheria.* The false membrane may be limited to a small patch on one or both tonsils, but in severe cases it may extend widely over a large area including both tonsils, the pillars of the fauces, the uvula, the soft palate and the posterior wall of the pharynx. The patient complains of a sore throat, headache and loss of appetite; fever is usually slight. In severe cases circulatory failure develops at an early stage and may cause death within a week or 10 days of the onset of the disease. Albuminuria is common and usually appears during the second week. Paralysis occurs in about 20 per cent of cases; it usually develops about the end of the third

or beginning of the fourth week. It most commonly involves the palate, the ocular muscles and the extensors of the wrists.

Faucial diphtheria has to be distinguished from tonsillitis and severe Vincent's angina. In advanced cases the characteristic appearance of the membrane and the general symptoms and signs leave no room for doubt, but in early cases diagnosis on clinical grounds alone may be impossible; *a swab should therefore always be taken for bacteriological examination by direct smear, culture and, in some cases, animal inoculation.*

*Laryngeal diphtheria.* Laryngeal diphtheria occurs almost exclusively in children and is usually caused by spread of infection from the fauces. The initial symptoms are hoarseness and croupy cough; later, severe respiratory obstruction may develop.

### **Treatment**

Subject to the precautions and limitations described on page 59, diphtheria antitoxin is given in doses of 10,000 to 50,000 units by intramuscular or intravenous injection *as soon as the disease is suspected on clinical grounds.* If the diagnosis is confirmed bacteriologically the injection of serum may be repeated. The mortality is negligible if serum is given on the first day of the disease but delay often proves fatal.

In addition to serum, penicillin injections may be given; these are effective against the organisms, but not against the toxin.

In laryngeal diphtheria intubation of the larynx or tracheostomy may be required to relieve respiratory obstruction.

### **Diphtheria prophylaxis**

The disease can be prevented by active immunization with diphtheria toxoid. The procedure is simple and should be carried out in early childhood; it has saved many thousands of lives.

Active immunity develops slowly; anyone in contact with a case of diphtheria who has not previously been actively immunized should therefore be passively immunized with antitoxin.

### **Anthrax**

Anthrax is caused by a large, aerobic, sporing, gram-positive bacillus (*B. Anthracis*), which occurs in long chains and possesses a well defined capsule. Infection is transmitted to man mainly from animal hides, wool or hair (for example, shaving brushes). Three forms of anthrax occur: cutaneous, pulmonary and intestinal.

**Cutaneous anthrax**

There is an incubation period varying from a few hours to a week. A red pimple then appears which is itchy but painless. Vesicles containing blood stained serum appear at the periphery and the centre becomes black and necrotic. Round the lesion—which is called a ‘malignant pustule’, though it contains no pus—the tissues become oedematous, and in the neck region oedema may spread to the larynx and cause respiratory obstruction. The related lymph nodes become enlarged and tender. In severe cases the temperature rises to 102° or 103° F. by the fifth day, the pulse becomes correspondingly rapid, vomiting may occur, and collapse and death may follow; in these cases there is usually an anthrax septicaemia. Fortunately the disease is usually less severe, and if proper treatment is given healing occurs in a few days.

*Diagnosis*

The condition must be distinguished from staphylococcal infections and accidental vaccinations (for example, lesions due to the vaccinia virus caused by contact with someone who has been recently vaccinated). The diagnosis is clinched by demonstrating the bacilli in fluid from the vesicles.

*Treatment*

Administration of penicillin in full dosage is usually effective, but in severe cases the patient may also be given an intravenous injection of specific antiserum.

The local lesion is kept clean and dry, but attempts at excision or cauterization are inadvisable.

**Other forms**

Pulmonary anthrax, also known as wool-sorter’s disease, is a fatal pneumonic condition. Intestinal anthrax is a serious and usually fatal condition characterized by colic, diarrhoea and the passage of blood per rectum.

**Tetanus**

Tetanus results from infection of a wound with the *Clostridium Tetani*, a gram-positive, sporing, anaerobic bacillus. The symptoms are due to an exotoxin formed by the bacilli which is absorbed by the end plates of motor nerves; it apparently travels via the nerves to the anterior horn cells of the spinal cord and thence, in severe cases, to the vital centres in the medulla.

The wound is usually either a small puncture wound or a large contused wound in which there is considerable damage to muscle. The organisms, usually in the form of spores, occur in soil and horse manure, and sometimes also in house dust. Occasionally they are introduced into the body during operations in improperly sterilized catgut.

The usual incubation period is from 4 days to a month, but the bacilli may remain latent in the tissues for years.

There are vague prodromal symptoms quickly followed by characteristic muscle spasms. The spasms may affect only the muscles in the neighbourhood of the wound, but usually occur also in other groups, including:

1. The muscles of the jaw causing *trismus*.
2. A muscle at the angle of the mouth causing '*risus sardonicus*'.
3. Muscles of the neck causing *neck stiffness*.
4. Muscles of the trunk causing various characteristic postures. The most striking is that known as *opisthotonus*, in which the patient's back is arched to such an extent that he virtually rests on his head and his heels.
5. In the late stages the *respiratory muscles* may be involved.

The spasms are intensely painful, and if they are untreated exhaustion and death may occur in a few days. In some severe cases the temperature may rise to 110° F., but often the temperature remains practically normal.

### **Diagnosis**

Tetanus must be distinguished from other conditions associated with muscle spasm, including trismus of dental origin, strychnine poisoning and hysteria.

Trismus due to dental conditions is discussed in Chapter XX. It can usually be recognized by finding evidence of local disease in the mouth and by the absence of other clinical features of tetanus. Strychnine poisoning causes painful tonic spasms, but the muscles relax completely between the spasms whereas in tetanus this never happens. Opisthotonus sometimes occurs as a manifestation of hysteria (Chapter XXI), but other signs of tetanus are absent and the spasms can often be controlled by suggestion.

The diagnosis is clinched by finding the characteristic bacilli in material obtained from the wound.

### **Prophylaxis**

Active immunization with tetanus toxoid confers a very high level of protection against subsequent infection with tetanus. It is a simple and safe procedure which should be undertaken routinely in everyone, and is especially important in gardeners, farm workers, those handling rubbish

and sewage, and those who undertake active sports in which injuries are common. The protection probably lasts for at least five years and if a 'booster' dose is given may last for twenty years.

Until recently it was customary in patients with abrasions, lacerations and penetrating wounds who had not been actively immunized previously, to undertake passive immunization by injecting 1500 units of antitetanic serum (ATS). This is still generally recommended in the patients with heavily contaminated wounds who are not seen until some hours after injury, subject to the precautions and limitations already described (p. 59). In addition the wound is excised and the patient is given 0.5 ml. adsorbed tetanus toxoid, and a course of penicillin for a period of three weeks. In other cases many surgeons now rely on penicillin and toxoid, combined sometimes with wound excision, and do not give ATS. Some, however, still prefer to give ATS routinely provided there is no contraindication. In patients who have previously received ATS, toxoid should also be given, however, because the ATS will be quickly eliminated.

### Treatment

Antitetanic serum is given, usually in a dose of 50,000 units, half intramuscularly and half intravenously, with the usual precautions. At one time much larger doses were used, but it is now realized that it is futile to try to neutralize toxin which has already become fixed to nervous tissues, and that the aim should be simply to neutralize all further toxin produced by the organisms. Local treatment of the wound is postponed for 1 to 3 hours after the first dose of serum has been given. The patient is nursed in a quiet dark room and sedatives are given to control the spasms, usually in the form of intravenous thiopentone. If this does not suffice muscle relaxants are administered under the supervision of an anaesthetist. Tracheostomy, with controlled respiration, is desirable in all but the mildest cases, and is essential if relaxants are used. Fluids, electrolytes and glucose are given intravenously and as soon as possible a high-calorie fluid diet is given by intragastric tube. Penicillin is given in full dosage by intramuscular injection.

### Gas Gangrene

Gas gangrene results from infection of wounds with a group of gram-positive, sporing, anaerobic bacilli, of which the *Clostridium Welchii* is the most important. The organisms are commonly introduced by contamination of the wound with cultivated soil. Infection is likely to become



established when there has been extensive laceration of muscles or when the circulation is impaired by haemorrhage, arterial injury, tourniquets, etc.

The infection spreads longitudinally in the muscles, causing them to become brick red and later black. Foul gas forms and gives rise to swelling of the part. At first the gas can be demonstrated only by radiological examination, but after a few hours *crepitus* (Chapter I) may be demonstrable. When the wound is opened up a watery offensive discharge escapes. There is profound toxæmia, resulting in nausea and vomiting, a rapid pulse, a high temperature which may fall shortly before death and a low blood pressure. The skin appears pale or cyanosed and the extremities are cold. In the terminal stages organisms may invade the blood stream and cause septicaemia.

### Treatment

The most important prophylactic measure is to perform thorough debridement of all contaminated wounds as soon as possible.

If gas gangrene develops the wound must be widely opened and irrigated with hydrogen peroxide, and the affected muscles excised; alternatively, in severe cases, amputation of a limb may be necessary. Penicillin is given by intramuscular injection in all cases. Anti-gas-gangrene serum is still used sometimes, but is of doubtful value.

### Actinomycosis

Actinomycosis in man is due to infection with the *Actinomyces Israeli*. This organism, which is sometimes called the 'ray fungus', forms masses of long branched threads (*hyphae*) at the periphery of which radiating club-like structures occur. It is gram-positive and anaerobic.

The source of infection has been much disputed. It used to be taught that actinomycosis was caused by chewing grass or sucking infected straw, but the organism commonly found on grasses or in soil is aerobic and non-pathogenic to man. Further investigation has shown that the *A. Israeli* is a common inhabitant of the mouth, and may gain entrance to the tissues through abrasions of the mucous membrane or after operative procedures including dental extraction.

Actinomycosis occurs mainly in the face and neck (Fig. 38) and in the gut (especially the region of the caecum), from whence it may spread to the liver. It occasionally affects the lungs and other parts of the body.



FIG. 38. Multiple sinuses due to actinomycosis.

#### *Cervico-facial actinomycosis*

In the neck a subcutaneous thickening develops, usually in the region of the angle of the jaw; this gradually enlarges and becomes markedly indurated. In the majority of cases the condition is painless. The infection spreads by direct continuity; it causes a marked fibrotic reaction, but the related lymph nodes do not normally show any change. After a time areas of softening appear and sinuses form; the sinuses discharge pus containing so-called 'sulphur granules' in which the organism can be demonstrated microscopically. The skin between the sinuses takes on a dusky colour.

In many cases there is associated dental sepsis or a history of recent dental extraction.

#### **Treatment**

Many strains of *A. Israeli* are sensitive to penicillin, and this drug should be tried in every case. Large doses should be given (for example, 1,000,000 units every 4 hours), and continued, if necessary, for weeks or months. It is very difficult to grow the organism in culture, and unless this can be done it is not possible to perform a sensitivity test; it is necessary, therefore, to rely on a therapeutic trial. If the condition does not respond to penicillin it is worth trying tetracycline.

X-ray therapy is of great value and may be used in conjunction with the antibiotics mentioned.

Administration of iodine or potassium iodide, once widely used, is becoming obsolete, but may be worth trying in intractable cases.

# Chapter XIII

## SPECIFIC INFECTIOUS DISEASES—II

### TUBERCULOSIS AND SYPHILIS

There are four varieties of the tubercle bacillus, of which two—the ‘human’ and the ‘bovine’—are pathogenic to man. Until recently bovine infections, though rare in adults, accounted for many cases of tuberculosis of the cervical or mesenteric lymph nodes and of bones and joints in children. Now, fortunately, in Britain and many other countries, milk is usually obtained from herds of cows which are free of tuberculosis, and in addition it is subjected to the further safeguard of pasteurization; in consequence the incidence of infection due to bovine bacilli in man has dropped sharply.

Though they differ considerably in their effects the human and bovine organisms are very similar in appearance. Both are gram-positive, but they are best stained by a special method—that of Ziehl-Neelsen—which demonstrates their acid-fast character. They are difficult to culture, but can be grown on certain special media.

Infection occurs by inhalation, ingestion or, occasionally, through a breach in the skin.

From the point of entry organisms may enter the lymphatic system and be carried rapidly to the regional nodes, or they may remain more or less localized. In either case *tubercles* are formed, consisting of giant cells, macrophages and lymphocytes, surrounding a centre of necrotic tissue. The necrosis is due partly to toxins produced by the organisms, and partly to progressive impairment of the blood supply to the centre of the tubercle resulting from the increasing accumulation of cells. The necrotic material has a cheeselike appearance and consistence; it is therefore described as *caseous*, and the process of necrosis in tuberculosis is termed *caseation*.

Subsequent events depend on the site of the lesion, the number and virulence of the organisms, the resistance of the patient and on whether or not the patient has been sensitized by previous tuberculous infection. The possible sequelae may be summarized as follows:

1. Healing of the lesion with the formation of a small scar.
2. The formation of a large amount of fibrous tissue.
3. Extensive caseation.
4. Cavitation, due to the discharge of caseous material (for example, its expectoration following ulceration of a pulmonary lesion into a bronchus) together with the formation of fibrous tissue.

5. The formation of a 'cold abscess' due to the liquefaction of caseous material. Such an 'abscess' contains a thick yellowish fluid which looks like ordinary pus, but which microscopically is seen to consist of fatty material and some round cells, but very few polymorphs.

6. The formation of a large amount of tuberculous granulation tissue, consisting of inflammatory cells (mainly macrophages and lymphocytes), fibroblasts and new capillaries.

7. Spread of infection to other parts of the body by direct continuity, or via lymphatics, epithelial-lined tubes, cavities such as the serous cavities and the subarachnoid space, or the blood stream (cf. Chapter IV).

Spread via the blood stream is of particular importance. It may lead to the development of one or two new localized foci in distant organs or, less commonly, to the condition known as *acute miliary tuberculosis* in which numerous small tubercles develop in various parts of the body, especially throughout the lungs, in the meninges and in the kidneys. Until recently acute miliary tuberculosis was almost always fatal but with modern chemotherapy 80 per cent of patients recover completely.

Individuals living in crowded cities usually become infected with tuberculosis in infancy or early childhood. From a pathological point of view the resulting 'primary' lesions have three important features:

1. There is relatively little tissue destruction; hence complete healing commonly occurs.

2. There is usually considerable enlargement of the related lymph nodes.

3. Occasionally acute miliary spread occurs.

Clinically, apart from the rare cases in which miliary tuberculosis supervenes, the condition usually runs a mild course and may pass unnoticed.

Most tuberculous lesions in older children and adults are due to reinfection. The pathological changes differ from those seen in primary lesions because the individual, having become sensitized or 'allergic' to the tubercle bacillus as a result of his previous infection, reacts differently. Tissue destruction and consequent fibrosis are far more extensive, but the regional lymph nodes are not enlarged to the same extent and miliary spread is rare.

### **Principles of treatment of tuberculosis**

Before describing the clinical features of tuberculosis in different sites it will be convenient to consider the principles which govern the treatment of tuberculosis in general.

*General treatment*

Chemotherapy, which began as an adjunct to other forms of treatment, is now the dominant factor. The main reason for this change is that the risk of the organisms becoming resistant, which was very great when streptomycin was used alone, is now very small if proper combinations of two or three drugs are used.

The three standard drugs are streptomycin (Chapter XI), isoniazid and para-amino salicylic acid (PAS). Isoniazid and PAS are synthetic drugs which are given orally.

When a patient has been treated inefficiently, due to faulty prescribing or failure of the patient to take the drugs prescribed, the infection may have become resistant to any combination of the standard drugs. The outlook is then much less hopeful but other drugs, including cycloserine and the tetracyclines, may be of value.

Since the initial treatment of a tuberculous patient is likely to be of decisive importance expert advice should always be sought before any specific drugs are given.

Ancillary general methods of treatment include rest, good food, fresh air and sunlight, and the treatment of any associated disorder such as anaemia.

*Local treatment*

Local treatment may take various forms, namely:

1. Treatment to provide local rest. A limb may be splinted or various types of operation may be performed such as arthrodesis (Chapter II) of a tuberculous joint.
2. Complete removal of a local lesion by operation; for instance, removal of a group of lymph nodes or part of a lung. Nephrectomy, which was once routine treatment for tuberculosis of the kidney, is now reserved for cases in which one kidney is extensively destroyed.
3. Curettage of a tuberculous focus where complete removal is impossible.
4. Aspiration of a cold abscess.

**Common sites for tuberculous lesions**

In miliary tuberculosis any part of the body may be affected and, as already mentioned, tuberculous meningitis is a serious complication.

Localized tuberculous lesions may also occur almost anywhere, but the common sites may be grouped together as follows:

1. Respiratory tract.
  - (a) Lungs.
  - (b) Larynx.

2. Lymph nodes.
3. Bones and joints.
4. Alimentary canal.
5. Kidneys, bladder and male genital organs.
6. Female genital organs.
7. Peritoneal cavity.
8. Skin.

From the point of view of this book tuberculous lesions of the cervical lymph nodes, bones and joints, and the skin are of especial interest and will therefore be discussed in detail.

### **Tuberculous cervical lymphadenitis**

Tuberculous cervical lymphadenitis is now quite rare, but before tuberculin testing of cattle and pasteurization of milk were widely practised it occurred commonly, especially in children, as the result of organisms entering the tissues from the mouth or pharynx, usually via septic tonsils. In a typical case the nodes soon become enlarged, but for some time remain smooth, firm and discrete. Later, caseation occurs and groups of glands become matted together as a result of periadenitis. Finally, the caseous material may become liquefied with the formation of tuberculous pus, or true pus if secondary pyogenic infection is present. In some cases the nodes become adherent to the overlying skin; the skin then becomes bluish-red in colour and eventually breaks down so that a sinus is formed. Sometimes a 'collar stud' abscess develops; that is, a collection of tuberculous pus in the subcutaneous tissues, communicating through a small opening in the deep cervical fascia with a caseous lymph node.

The nodes mainly involved are those of the jugular chain, especially the jugulo-digastric group situated near the point where the posterior belly of the digastric muscle crosses the internal jugular vein (Figs. 39 and 40).

### *Treatment*

Standard antituberculous chemotherapy is given using an appropriate combination of drugs (*v. supra*). Some cases respond well in the course of a few weeks; others fail to respond and in this event, if the condition is localized to one or two groups of nodes, these should be excised *before* the development of periadenitis and caseation makes this difficult. In either case chemotherapy should be continued for at least a year. If the patient is not seen until the later stages a small incision is made through which liquefied material is evacuated and breaking-down glands are curetted. This

incision is sutured round a small drain which is removed after 24 hours. In suitable cases excision of the scar and any remaining nodes is undertaken subsequently.



FIG. 39



FIG. 40

FIG. 39. Enlargement of the jugulo-digastric nodes due to tuberculous lymphadenitis.

FIG. 40. Tuberculous lymphadenitis in an infant.

If there is widespread involvement of the cervical lymph nodes an X-ray of the chest often reveals enlarged nodes in the mediastinum. Complete surgical excision is impossible and prolonged conservative treatment is required.

### **Tuberculosis of bones and joints**

Tubercle bacilli may be carried by the blood stream from a focus elsewhere in the body and set up infection in a bone or joint. The primary focus, which may or may not be demonstrable, is usually situated in a cervical or mesenteric lymph node.

Infection may spread from a bone to a nearby joint—for example, from the head of the femur to the hip joint; but tuberculosis in a rib or in one of the small bones of the hand, without any joint involvement, also occurs.

Conversely, if the synovial membrane of a joint is infected via the blood-stream, involvement of the ends of the bones taking part in the articulation usually occurs rapidly. Occasionally, however, this is not the case; tuberculosis of the knee joint, for example, usually begins as a purely synovial infection and the bones may remain uninfected for a considerable time.

Tuberculous arthritis is most commonly seen in children, but sometimes develops during adult life.

*Morbid anatomy*

The synovial membrane shows scattered tubercles, masses of tuberculous granulation tissue or, in some cases, extensive caseation. The articular cartilage gradually becomes eroded and replaced by granulation tissue.

There is usually considerable destruction of bone with caseation, but little or no new bone formation. The muscles which move the joint become wasted. In the late stages a cold abscess may form and subsequently discharge on the surface through one or more sinuses.

The commonest end result is *fibrous ankylosis*; that is, fixity of the joint due to fibrous tissue (Chapter XX). Bony ankylosis very rarely occurs, except as a result of either surgical operations deliberately undertaken with this end in view or secondary pyogenic infection. Sometimes the joint becomes so seriously disorganized that pathological dislocation occurs.

*Clinical features*

The onset is usually insidious. There is rarely much pain in the early stages, except sometimes for 'night pains'. These occur just as the patient falls asleep. The muscles relax allowing the articular surfaces to rub against each other and the patient wakes suddenly with a cry. Later, when erosion of cartilage has occurred, pain may be severe.

There is usually some swelling of the joint, and movement soon becomes limited in all directions owing to muscle spasm. Muscular wasting is usually marked (Fig. 41). There may be general symptoms and signs such as

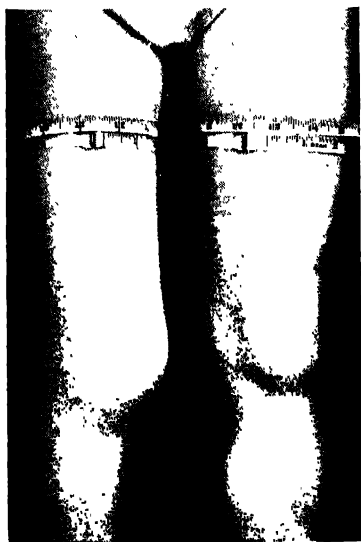


FIG. 41. Tuberculosis of the right knee joint with wasting of the quadriceps muscle. The normal left knee is shown for comparison.



listlessness and mild pyrexia. In the later stages tuberculous sinuses and deformity due to pathological dislocation may be seen.

X-ray examination in the early stages shows either nothing abnormal or slight general decalcification of the bones. Later, areas of bone destruction can be seen and the joint space may appear narrowed owing to destruction of the articular cartilage (Fig. 42).

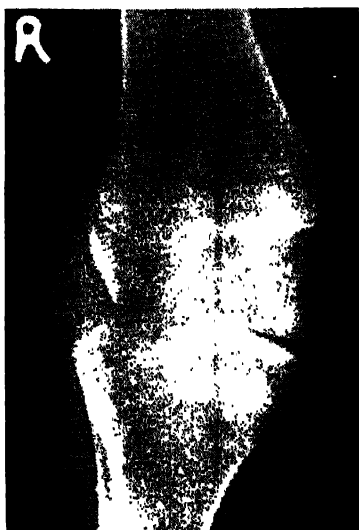


FIG. 42. *A.* Radiograph of a tuberculous knee joint in an adolescent.  
*B.* The opposite normal joint for comparison.

### *Treatment*

General treatment, including standard chemotherapy, along the lines already described should be instituted in all cases.

Local treatment may be entirely conservative or partly operative. Conservative treatment consists essentially in immobilization by splinting until the infection is completely quiescent and fibrous ankylosis of the joint has occurred.

Operative treatment is undertaken in some cases, either to shorten the period of treatment or to increase the chance of permanent cure. The commonest type of operation is *arthrodesis*—that is, the production of firm bony ankylosis of the joint, but sometimes *arthroplasty*, which is designed to provide a mobile joint, is feasible.

### **Tuberculous dermal lesions**

Tuberculous dermal lesions are of three main types:

1. *Ulcers or sinuses related to a deep-seated tuberculous focus*, usually in a bone, joint or lymph node. The treatment is directed to the underlying lesion.

2. *Anatomical tubercle or 'butcher's wart'*. This condition is due to direct infection of the skin with tubercle bacilli. Intact healthy skin is extremely resistant to such infection, but the organisms may gain entrance through small cracks or abrasions. The condition is rare and is seen mainly in pathologists, post-mortem room attendants and butchers.

The initial lesion is a small indurated papule; this develops into a hard wart-like mass. The related lymph nodes soon become enlarged and may later break down. Excision of the primary lesion is usually advised; this should be combined with the usual general treatment.

3. *Lupus vulgaris*. This is a condition which occurs most commonly in the region of mucocutaneous junctions, but is occasionally found elsewhere. Both children and adults may be affected.

In the usual type the lesions appear first on the mucosa of the lips or nose and spread to the skin. Small intradermal nodules develop which, if a glass slide is pressed against the skin, appear pinkish and semi-translucent like apple jelly. The lesion spreads in a centrifugal manner, fresh nodules appearing at the periphery while scarring occurs in the centre (Fig. 43).



FIG. 43. *Lupus vulgaris*.

The condition is disfiguring but rarely dangerous. Occasionally, however, an epithelioma develops at the site of an old lupus scar.

Treatment consists in standard antituberculous chemotherapy together with administration of large doses of vitamin D (Calciferol). When disfigurement has occurred the patient's appearance can sometimes be improved by providing a suitable prosthesis (Fig. 44).

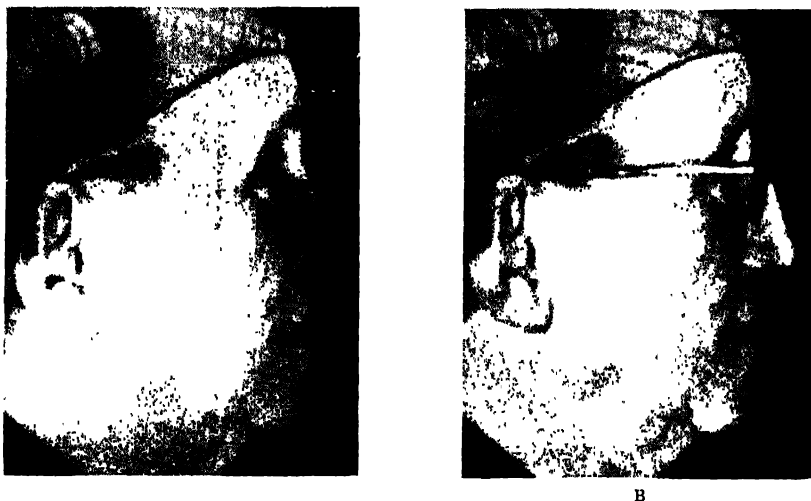


FIG. 44. A. Destruction of nose due to lupus.  
B. Plastic prosthesis attached to a spectacle frame.

## Syphilis

Syphilis is an infectious disease due to the *spirochaeta pallida*. The infection may be acquired or congenital.

### Acquired syphilis

#### *The primary lesion*

The primary lesion is termed a *chancre* (Fig. 45). It appears 2 to 6 weeks after infection as a small reddish papule which soon breaks down to form a hard and painless ulcer. The edge of the ulcer is somewhat heaped up and slopes down to the floor, which is red or yellow in colour and oozes thin serum containing numerous spirochaetes. The related lymph nodes are enlarged and hard, but remain discrete and are not tender.

The chancre is usually situated on some portion of the external genitalia, but extra-genital chancres occur occasionally on the lips, tongue, inside of the mouth, breast or abdominal wall.

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The histological features are those characteristic of syphilitic lesions in general; notably, accumulation of lymphocytes, plasma cells and some macrophages; proliferation of fibroblasts; and endarteritis of small vessels.

Even if it is untreated the primary sore heals in a few weeks unless there is marked secondary pyogenic infection.

### Secondary lesions

Two to 4 months after infection lesions appear on the skin and mucous membranes; the disease is then said to have reached the secondary stage.

The skin shows a red macular rash (Fig. 46) which deepens to a tint described as 'copper coloured'. A papular eruption also appears in many



FIG. 45

FIG. 45. A chancre on the deep surface of the prepuce.

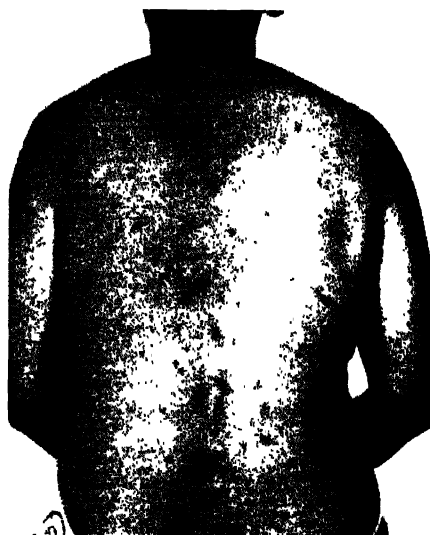


FIG. 46

FIG. 46. Rash due to secondary syphilis.

cases. These rashes commonly occur over most of the body. The appendages of the skin are also affected, the hair being lost in irregular patches, and the nails becoming brittle and fissured. Sometimes there is scab formation with extensive underlying tissue destruction; the resulting lesions resemble brown limpet shells and are known as *rupia*. In moist situations wart-like excrescences known as *condylomata* appear.

Local symptoms of irritation such as pain and itching are uncommon, but there may be mild general symptoms as described below. Owing to the presence of spirochaetes in large numbers the lesions are highly infectious.

*Mucous membranes.* The characteristic lesion is the *mucous patch*, an area of sodden thickened epithelium which rapidly breaks down to form a very

superficial ulcer covered with mucoid exudate ('*snail track ulcer*'). These lesions occur mainly, but not exclusively, in the mouth and throat. Like the skin lesions they are highly infective.

*General symptoms and signs.* There may be headache, mild fever, secondary anaemia, general enlargement of lymph nodes, and aching pains, especially in the bones.

Like the primary chancre the secondary manifestations disappear completely even without treatment; spirochaetes lie dormant in the body, however, and after a varying period—from months to years—tertiary lesions appear. During the period between the disappearance of the secondary lesions and the appearance of tertiary lesions the disease is said to be *latent*.

### *Tertiary lesions*

Tertiary lesions are of two main types:

1. Gummata.
2. Diffuse syphilitic inflammation with fibrosis.

A *gumma* is a mass of necrotic tissue with surrounding syphilitic inflammation. The dead material is firm and resilient, quite unlike the caseous type of necrotic material seen in tuberculosis; this difference depends on differences in the enzymes liberated by the spirochaete on the one hand and the tubercle bacillus on the other.



FIG. 47. Gummatous ulcers.

When a gumma is situated near to the surface ulceration sooner or later occurs. The resulting ulcer presents a characteristic appearance (Fig. 47), being roughly circular with a clear-cut 'punched out' edge and a sloughing floor (the so-called '*wash-leather slough*').

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Gummata may occur in the skin, bones, nervous system or viscera. They are usually painless.

*Diffuse syphilitic infiltration* is seen most commonly in the cardiovascular system, the nervous system including the meninges, the liver, testes, and bones. There is gross destruction of specialized tissue with ensuing fibrosis or, in the case of bone, sclerosis. When the wall of a large blood vessel—most commonly the aorta—is involved, an *aneurysm* (Chapter XVII) is liable to form.

*General paralysis of the insane* and *tabes dorsalis*—two nervous diseases sometimes referred to as *parasyphilis*—are due to diffuse syphilitic infiltration of the brain and the posterior columns of the spinal cord respectively. *Tabes dorsalis* has some surgical significance in that it leads to anaesthesia of deep structures such as tendons, muscles and joints, and may result in the development of painless but grossly disorganized joints, known as *Charcot's joints* (Fig. 48).



FIG. 48. Radiograph of Charcot knee joint.

**Congenital syphilis**

A syphilitic mother usually has a series of abortions or still births before giving birth to a live child. At birth this child may appear normal, but after about a month evidence of congenital syphilis commonly appears. The child loses weight, becomes irritable and anaemic, and often vomits. The face becomes wrinkled like that of an old man, and the spleen and liver are usually found to be enlarged. Lesions resembling those of the secondary stage of acquired syphilis soon appear. Ulceration of the nasal mucosa leads to a constant discharge known as 'snuffles', and spread of infection to the bones of the nose causes the bridge to fall in, giving a characteristic appearance. Cracks commonly appear at the angles of the mouth (Fig. 49); this condition is known as *rhagades*.



FIG. 49. Child with congenital syphilis, showing rhagades and depression of the bridge of the nose.

*Syphilitic otitis media* may occur by spread of infection along the Eustachian tube from the nose.

If untreated the infant usually dies, but if it survives other manifestations develop.

The earliest of these later manifestations are *syphilitic epiphysitis* and *periostitis*. On the skull periostitis results in the development of bosses known as *Parrot's nodes*; these give the skull a characteristic appearance which has been likened to that of a hot cross bun. Defects in the skull, due to replacement of bone by a thin membrane, also occur; this condition is known as *cranio-tabes*.

Still later—usually between the fifth year and puberty—the child is liable to become lame, deaf, blind and potentially impotent; *lame* because of the development of changes in the bones and joints ('sabre tibia' and 'Clutton's joints'); *blind* because of interstitial keratitis and choroiditis; *deaf* because of syphilitic otitis interna; and *potentially impotent* because of syphilitic orchitis.

The teeth of the second dentition may show characteristic changes. The first molars may become dome-shaped (*Moon's molars*), and the upper central incisors are frequently peg-shaped (that is, broader near the gums than at the cutting surface) and sometimes notched (*Hutchinson's teeth*—Fig. 50). Notched teeth may also occur as a result of rickets, but the notch then takes the form of a small arc of a large circle whereas in congenital syphilis it is usually in the form of a large arc of a small circle.



FIG. 50. Hutchinson's teeth.

### Diagnosis of syphilis

In addition to the clinical features there are two special types of investigation of great diagnostic importance:

1. *Microscopic examination of smears for spirochaetes using dark ground illumination.*

This is of importance in diagnosing primary and secondary lesions.

2. *Serological investigations*, of which the Wassermann complement fixation test and the Kahn test are the most generally useful.

### Treatment of syphilis

Penicillin has revolutionized the treatment of syphilis and may be used at any stage of the disease. In primary and early secondary cases, for instance,



a permanent cure can usually be obtained by treatment for 2 weeks or less, provided large doses are used.

In late syphilis even larger doses are used, preceded sometimes by a course of intramuscular injections of bismuth.

Organic arsenical preparations such as neoarsphenamine, which were once of great importance, are now used only in patients who are intolerant of penicillin.

## *Chapter XIV*

# TUMOURS

The term 'tumour' is sometimes used clinically to mean any kind of localized swelling. Strictly speaking, however, it should only be used to denote new growths or 'neoplasms'.

What is a neoplasm? No one has yet succeeded in giving a completely satisfactory answer to this question. Handfield-Jones defines a neoplasm as 'a mass of cells, tissues or organs resembling those normally present in the body, but arranged atypically, which grow at the expense and independently of the organism, but without subserving any useful purpose therein'. For want of a better this must serve as our definition; it is, however, open to criticism, because no tumours are really independent of the organism in which they grow, and some are greatly influenced by environmental factors including, in particular, hormonal stimulation.

The difficulty of definition is not primarily a linguistic one, but occurs because we have so little knowledge of the fundamental nature and causation of the abnormal growth we speak of clinically as neoplastic. Despite the enormous amount of research which has been undertaken into the origin of tumours who can say, for instance, why one patient develops a cerebral tumour and another does not, or why carcinoma is common in the stomach but almost unheard of in the duodenum?

Some advance, however, has been made. In the first place, it is now recognized that there is no single cause which is responsible for all neoplasms. Secondly, one can point, in some cases, to the operation of specific etiological factors such as hereditary predisposition, chronic mechanical or chemical irritation, syphilitic infection or hormonal stimulation. Filtrable viruses might perhaps be added to this list; they are responsible for some experimental tumours but have not so far been proved to play any part in the production of tumours in man.

Neoplasms are subdivided into two main groups: benign and malignant. The distinction between the two is fairly clear-cut and most neoplasms fall into one or other group, but there are a number of border-line cases. Occasionally a benign tumour undergoes malignant change, but this is less common than used to be supposed.

**Benign neoplasms**

Benign growths are not dangerous to life unless, by virtue of their position, they interfere with the function of vital structures. A benign neoplasm within the cranial cavity, for instance, may cause death by mechanical compression of the brain.

Histologically, benign neoplasms resemble very closely the ordinary tissues of the body. They may grow to considerable size, but remain walled off by a capsule of connective tissue and merely displace the surrounding tissues without invading them. Clinically they form well-defined swellings which are not attached to neighbouring structures. They do not spread to other parts of the body, and unless there are exceptional effects due to pressure the general health of the patient is unaffected.

Various types of benign neoplasm are described depending on the nature of the tissue from which they arise. They may be classified as follows:

1. *Epithelial growths*. These are further subdivided into *papillomata* originating in surface epithelium and *adenomata* originating in glandular epithelium. In both cases the neoplastic tissue is epithelial, but there is a supporting meshwork or 'stroma' of connective tissue containing blood vessels and nerve fibres.

2. *Non-epithelial neoplasms*. These include lipomata, fibromata, osteomata, chondromata, osteoclastomata or giant-cell tumours, fibromyomata, neurofibromata and many other less common types.

3. *Melanomata* or pigmented moles. These contain a pigment known as melanin. They are placed in a special category because it is not certain whether the pigment-bearing cells of the skin from which they are derived originate from epidermal cells or, as the work of Masson suggests, from cells which have migrated to the skin from the neural crest during embryonic development.

4. *Teratomata*; that is, neoplasms containing both epithelial and mesoblastic elements. Benign teratomata are rare, but occur occasionally especially in the ovary.

**Principles of treatment**

In some cases no treatment is necessary. If, however, the growth is causing pressure effects or is unsightly, or if there is doubt about its pathological nature, it should be excised; alternatively, in some cases, it may be destroyed by diathermy or other means. Radiotherapy (*vide infra*) is *not* indicated except in the special case of osteoclastomata (Chapter XIX).

Complete removal of a benign tumour will effect a permanent cure, but if portions are left behind recurrence may occur.

### Malignant Neoplasms

Malignant neoplasms cause progressive deterioration in health, and ultimately death, unless they are removed or treated by radiotherapy at an early stage. The cause of their deleterious effect is obscure. It appears to be due in part to the fact that a malignant tumour is able to abstract from the blood stream an excessive amount of nutrient material, so that the patient may be virtually starved. This is all the more serious because in many cases he suffers from *anorexia* (loss of appetite) so that his intake of food is severely restricted. A further factor of importance in some cases is secondary infection with consequent bacterial toxæmia.

Malignant tumours often increase rapidly in size and the surrounding tissues are infiltrated instead of being merely compressed. Wide excision is therefore essential if local recurrence is to be avoided.

In many cases in addition to local infiltration there is spread to distant parts of the body; this process is termed *metastasis*. Metastasis may occur by any of the routes described in Chapter IV for the spread of bacterial infection, but metastasis by lymphatics to the regional lymph nodes, and metastasis by the blood stream, are of special importance.

Metastasis by lymphatics may occur as a result of *permeation* (that is, continuous growth of tumour tissue along the vessels); or by *lymphatic embolism* (that is, transportation of detached tumour cells or small fragments of tumour tissue in the lymph stream).

Metastasis by the blood stream occurs as a result of embolism. If the primary growth is drained by the portal venous system the emboli are carried first to the liver; if by the systemic venous system they go to the lungs. Many go no further, but small emboli may succeed in traversing the capillary bed and then be carried from the liver to the lungs, or from the lungs, via the arterial blood stream, to any part of the body.

It has been shown that only a small proportion of the tumour emboli which become lodged in the capillaries of the liver, lungs and other organs survive and form metastatic growths. The factors which determine survival have not been elucidated, though various theories have been propounded. The matter is of great importance because if metastasis could be prevented malignant disease would lose many of its terrors.

Histologically, malignant neoplasms usually differ materially from normal body tissues, both as regards the individual cells and the manner of their arrangement. Sometimes it is extremely difficult or impossible to determine from the histological appearance the tissue of origin; the neoplasm is then termed *anaplastic* and is usually highly malignant.

Clinically, malignant neoplasms often have a somewhat indefinite edge and may be fixed to surrounding structures. Lymphatic or blood-borne metastasis may be apparent. There is often marked loss of weight, generalized muscular wasting and anaemia.

Malignant neoplasms may be classified as follows:

1. *Carcinomata*; that is, malignant neoplasms of epithelial origin.
  - (1) Arising from squamous epithelium.
    - (i) Basal-cell carcinoma, sometimes called rodent ulcer.
    - (ii) Squamous-cell carcinoma, sometimes called epithelioma.
  - (2) Arising from glandular epithelium.
    - (i) Adenocarcinoma.
    - (ii) Carcinoma simplex.

The term carcinoma simplex, though widely used, is not well chosen. Such growths are 'simple' in a histological sense because they contain masses of epithelial cells without the complex glandular formations characteristic of adenocarcinomata. Generally speaking, however, they carry a much less favourable prognosis than adenocarcinomata and the problems they present to the surgeon are the reverse of simple.

All carcinomata contain, in addition to the malignant epithelial cells, a 'stroma' of connective tissue, blood vessels and nerves.

2. *Sarcomata*; that is, malignant neoplasms of mesodermal origin. Sarcomata, unlike carcinomata, are not uncommon in children and adolescents, but may occur at any age. They may arise in connective tissue, cartilage, bone, muscle, fat and lymphoid tissue. They are usually highly malignant and blood-borne metastasis is common, especially to the lungs (Fig. 51).

Some sarcomata, like osteogenic sarcoma (Chapter XIX) which is the common primary malignant tumour of bone, show sufficient differentiation for the tissue of origin to be recognized histologically. Most, however, consist mainly of round cells, spindle-shaped cells or a mixture of the two; they are then termed respectively round-cell, spindle-cell and mixed-cell sarcomata. Lymphosarcoma is a highly anaplastic growth which originates in lymphoid tissue and differs from other sarcomata in several important respects. It will be described in Chapter XVIII.

3. *Malignant melanomata*.

4. *Malignant teratomata*.

The initial growth, of whatever type, is termed *primary*; growths which develop later as the result of metastasis are termed *secondary*.

The term *cancer* is often used as a synonym for malignant tumours in general.



FIG. 51. Radiograph showing multiple metastases in the lungs. The primary tumour was an osteogenic sarcoma.

### Principles of treatment

Malignant neoplasms may be treated by surgery, radiotherapy or chemotherapy, or by a combination of these procedures.

### Surgical treatment

Surgical treatment is usually best, provided that the following conditions are fulfilled.

1. The primary growth can be excised completely without endangering the life of the patient or producing gross disability or deformity.
2. There are no blood-borne metastases.
3. Either there are no lymphatic metastases or the affected nodes, together with the primary growth and the connecting lymphatic channels, can be removed *en bloc*.

At operation the primary growth is excised with a wide margin of healthy tissue; in addition, in many cases, an attempt is made to remove the related lymph nodes and lymphatic vessels, even if these show no obvious involvement, because metastasis may have occurred, even though it is not yet apparent.

Some reconstructive procedure may be necessary to complete the operation. After excision of a carcinoma of the lip, for instance, the margins of the V-shaped gap which is left must be brought together; after excision of a carcinoma of the colon the continuity of the bowel must be re-established or the proximal loop brought out as a *colostomy* (artificial anus).

### **Radiotherapy**

The term radiotherapy includes the following procedures:

1. Irradiation from an external source which is installed in a special room to which the patient is taken for treatment. The source may take the form of either (a) a conventional X-ray tube or a supervoltage machine such as a linear accelerator, or (b) a large mass of radioactive material, usually a radioactive isotope of cobalt ( $^{60}\text{Co}$ ).
2. Irradiation from radioactive material inserted into the tumour.
3. Administration of radioactive substance orally or by injection.

#### *Irradiation from an external source*

X-rays are a form of electromagnetic radiation having a wavelength very much shorter than that of light. Such radiation in sufficiently high dosage is lethal to all tissues, but some tumours are particularly *radio-sensitive*; that is, their cells may be destroyed by radiation in a dosage which is tolerated by normal tissues.

The higher the voltage at which X-rays are generated the shorter their wavelength and the greater their penetrating power. Conventional X-ray therapy machines are operated at voltages of 150 to 250 kilovolts. Supervoltage machines, which work on a somewhat different principle, generate radiation whose wavelength is equivalent to what would be produced in theory by conventional machines if they could be made to operate at 1 million or more volts. Supervoltage machines are very costly, but have the advantage that, because of the great penetrating power of the irradiation they produce, large doses can be given to deep-seated tumours with much less risk to the overlying tissues than is the case when a conventional 250 kilovolt machine is used.

A cobalt source provides an economical alternative to a supervoltage machine. The atoms of  $^{60}\text{Co}$ ., like those of other radioactive elements, are unstable and undergo spontaneously a complex series of changes which are associated with the emission of  $\alpha$  and  $\beta$  particles and  $\gamma$  rays.

It is the  $\gamma$  rays which are used therapeutically. They are physically similar to X-rays but because of their difference in origin have been given a different name.

A  $^{60}\text{Co}$ . source loses its activity fairly slowly. In technical language the 'half-life period' is 5 years. This means that after 5 years half of the atoms in the original source will have disintegrated and half will remain unchanged; after 10 years only a quarter will remain, and so on. Clearly, therefore, to maintain therapeutic efficiency the source should be replaced every few years.

X-rays and  $\gamma$  rays have the power of ionizing gases, and the *roentgen* (or *r* unit) is defined as the quantity of radiation that produces ions carrying 1 electrostatic unit of electricity of either sign in 1 ml. of air under standard conditions. The energy absorbed in air for 1 roentgen is 83 ergs per gram and this is approximately true also in the soft tissues of the body. The same does not hold good for bone, however, and for this reason therapeutic doses of irradiation are now usually expressed as *rads* rather than as roentgens, one rad being defined as the quantity of irradiation which corresponds to 100 ergs of absorbed energy in the tissue irradiated.

#### *Irradiation from substance buried in the tumour*

Some tumours may conveniently be treated by *interstitial irradiation*, i.e. by inserting into them radioactive material in the form of radium needles, radon seeds or radioactive cobalt or tungsten wire. Those procedures have been partly superseded by supervoltage X-ray therapy, but are still used in many clinics for the treatment of some tumours of the tongue, pharynx and bladder.

*Radium needles.* These are platinum tubes containing a small quantity (usually between 1 and 5 milligrammes) of one of the salts of radium, made gas-tight to prevent escape of radon. The walls permit the passage of  $\gamma$  rays but are sufficiently thick to block the harmful  $\alpha$  and  $\beta$  particles. As a rule the amount of radium used is such that the required dose of radiation will be delivered in about a week, and at the end of this time the needles are removed. The half-life of radium is extremely long (1,550 years) so that if the needles were not removed, the patient would continue to be irradiated, *with only a very slight diminution of intensity*, for the rest of his life.

*Radon seeds.* Radon is collected and used to fill small gold containers which are known as radon seeds. Like radium needles, these are gas-tight and sufficiently thick-walled to prevent the escape of  $\alpha$  and  $\beta$  particles.

The half-life period of radon is a little less than 4 days, so that if we start with a quantity of radon appropriate for therapeutic use the amount which remains after a few weeks is negligible. Since all the other  $\gamma$  ray



emitters which are formed during the subsequent transformation of radon also have a very short half-life period, radon seeds may safely be left in the body indefinitely. This is a great advantage when the tumour to be treated is relatively inaccessible.

*Cobalt and tungsten wire.* Radio-active cobalt or tungsten in the form of short lengths of wire is sometimes used as an alternative to radium needles.

#### *Administration of radio-active substance orally or by injection*

Radioactive iodine ( $^{131}\text{I}$ ) is used in the treatment of carcinoma of the thyroid. Unfortunately the value of this procedure is limited by the fact that as a rule these tumours do not take up iodine to anything like the same extent as normal thyroid tissue.

#### *Indications for radiotherapy*

Radiotherapy would be preferable to excision of a tumour, especially where excision would be severely mutilating, if it offered equally good prospects of cure. Does it ever do so? With tumours which are not radio-sensitive the question does not arise since radio-therapy is clearly useless, but in other cases there is wide disagreement about the relative efficacy of radiotherapy and surgical excision.

In Britain radiotherapy is used to a greater extent than in the United States, and is regarded by many surgeons as the treatment of choice for basal-cell carcinomata and for the primary lesion in most epitheliomata of the skin, lip, tongue, buccal cavity and *cervix uteri*.

Radiotherapy is also valuable as a palliative procedure for widespread inoperable tumours, with or without metastases, where no treatment offers any prospect of cure.

Generally speaking, radium needles (or other radio-active materials) are implanted when it is desired to deliver a large dose of radiation to a relatively small volume of tissue, and deep X-ray therapy is used when it is desired to treat a large volume of tissue, either in the hope of cure or as a palliative procedure.

The indications for using radon in preference to radium, have already been mentioned.

In this chapter we are primarily concerned with tumours, but it may be remarked in passing that radiotherapy is of value in a number of non-neoplastic conditions including actinomycosis (Chapter XII), parotid fistula (Chapter XVI), haemangiomas (Chapter XVII) and Paget's disease of bone (Chapter XIX).

### Chemotherapy

A great effort is being made to develop drugs which are effective for the treatment of cancer. Most of the agents which have been investigated fall under one of the following three headings: *hormones* of various kinds, *alkylating agents* such as nitrogen mustard, and *antimetabolites* like 6-mercapto-purine. So far chemotherapy offers little or no prospect of cure, but good palliation may be obtained with some advanced tumours.

### Combined methods

Many cases of malignant disease are best treated by a combination of surgery and radiotherapy. An epithelioma of the lip with secondary deposits in the cervical lymph nodes, for example, is usually treated by irradiation of the primary growth followed by surgical excision of the lymph nodes 2 or 3 weeks later.

We have dealt so far with the principles underlying the pathology, diagnosis and treatment of neoplastic disease. It now remains to describe in detail some tumours of special importance to the dental student.

## Tumours of the Skin

### Papilloma

Papillomata arising from the skin constitute one variety of wart. They may occur anywhere on the surface of the body, but are particularly common on the scalp. If unsightly or otherwise annoying to the patient they may be excised or destroyed by diathermy under local anaesthesia. Warts which are due to a virus infection, and are probably not true tumours, commonly occur on the hands. They may be treated as above or by application of carbon dioxide snow. Local application of silver nitrate and other caustics is often used, but is messy and unsatisfactory.

### Basal-cell carcinoma

Basal-cell carcinomata (rodent ulcers) arise from the deeper layers of the skin. They are of relatively low malignancy and spread very slowly until they invade bone, after which rapid extension may occur. Distant metastasis by lymphatics or the blood stream is unknown.

Rodent ulcer occurs most commonly on the face, especially just below the eye and on the side of the nose. The initial lesion is a small raised nodule, but this soon breaks down to form an ulcer (Fig. 52). The edge of the ulcer has a characteristic beaded appearance and the floor is usually



FIG. 52. Early basal-cell carcinoma.



FIG. 53. Hideous deformity resulting from a neglected basal-cell carcinoma.

covered by a scab. Secondary bacterial infection commonly occurs. If the condition is neglected hideous deformity may result (Fig. 53)

The important differential diagnosis is from squamous-cell carcinoma. The main points of difference are that rodent ulcer grows more slowly and shows much less heaping up of epithelium at its edge. Very occasionally a rodent ulcer changes its character and develops into a true squamous-cell carcinoma.

### *Treatment*

Rodent ulcers are almost always radio-sensitive, and early cases may be treated effectively by X-ray therapy; alternatively they may be excised. Extensive ulcers with severe sepsis are very difficult to treat.

### **Squamous-cell carcinoma**

Squamous-cell carcinoma of the skin begins as an indurated nodule which gradually increases in size and extends into the underlying tissues. Sooner or later the surface epithelium breaks down and an ulcer is formed (Fig. 54), having a raised everted edge and an indurated base. Metastasis to lymph nodes is common, but metastasis by the blood stream is extremely rare.



FIG. 54. Ulcerating squamous-cell carcinoma.

### *Treatment*

The primary lesion may be excised or treated by X-ray therapy. If excision is used the growth is removed with a surrounding area of healthy tissues and if the skin edges cannot be brought together a graft is used to cover the raw area.

If there is lymph-node metastasis a *block dissection* of the nodes must be performed. If, for instance, the primary growth is on the leg, and the inguinal nodes are involved, a dissection is made in the groin and all the nodes, together with the surrounding fat and connective tissue, are removed in one piece; if the primary growth is on the arm a similar operation may have to be performed in the axilla.

**Melanoma**

Benign melanomata (pigmented moles) occur commonly in the skin. They take the form of either small pedunculated tumours resembling papillomata or flat brown or black plaques (Fig. 55). Hairs sometimes grow exuberantly from these plaques and the lesion is then known as a *hairy mole*.



FIG. 55. A large pigmented mole in an infant.

Pigmented moles must be regarded with suspicion. The majority remain unchanged throughout life, but some undergo malignant change. This may be indicated clinically by bleeding or sudden increase in size, but even before these warnings occur widespread dissemination of the growth may have taken place. Malignant change is often initiated by trauma; for example, by inadequate attempts at removal or by repeated mild trauma such as occurs in a melanoma of the scalp from combing the hair.

Malignant melanomata commonly develop from pre-existing benign growths, but some appear to be malignant from the start. They form soft fleshy tumours which grow rapidly (Fig. 56). The degree of pigmentation varies greatly; some tumours are quite pale and contain very little melanin, others are jet black in colour.

Metastasis to lymph nodes and by the blood stream is common. Blood-borne metastases may occur anywhere, but are particularly common in the liver and subcutaneous tissues. The prognosis in malignant melanomata is extremely grave, especially as these tumours are rarely radio-sensitive.

***Treatment***

Pigmented moles which are not exposed to trauma and which show no evidence of malignant change are usually best left alone. Those in exposed

situations should be excised with a wide margin of healthy tissue, great care being taken to avoid injury to the actual growth.

Malignant melanomata which show no evidence of metastasis should also be excised. Some surgeons advocate block dissection of the regional lymph nodes at the same time, or an even more radical dissection in which an attempt is made to remove *en bloc* the primary growth, the nodes and the intervening lymphatic vessels. Such heroic operations are occasionally successful, but in many cases the patient returns within a few months with metastases in other lymph nodes or widely disseminated in the liver, lungs, skin and other tissues.

Radiotherapy is, unfortunately, rarely of value.



FIG. 56. Malignant melanoma of the great toe.

## Tumours of the Lips, Tongue and Mouth

### Benign tumours

The benign tumours which occur in the mouth are papillomata, 'mixed' tumours and epulides. Odontomes, haemangiomata and lymphangiomata

are sometimes included with benign tumours, but most of these are not true neoplasms; they will therefore be considered in other chapters.

### *Papilloma*

A papilloma occurs occasionally on the lips, tongue, gums, soft palate or inner surface of the cheeks. It forms a small pedunculated or wart-like swelling. The treatment is excision; this is usually done under local anaesthesia.

### *Mixed tumours*

A benign tumour, similar in structure to the mixed salivary tumour which occurs in the parotid gland (page 190), sometimes develops from one of the small mucous glands in the mouth. It occurs usually on the palate and forms a firm, lobulated swelling, covered with intact mucous membrane. The treatment is excision.

### *Epulis*

The term epulis is applied to two distinct types of tumour which occur in the mouth in close proximity to the teeth.

*The fibrous epulis* is a fibroma which arises from the periodontal membrane or alveolar periosteum. It forms a smooth or somewhat lobulated swelling on the outer aspect of the gum (Fig. 57), sometimes in relation



FIG. 57. Fibrous epulis. It is unusual for this tumour to occur in an edentulous patient.

to one or more carious teeth, especially the lower molars or premolars. The tumour has a broad pedicle but is usually mobile on the alveolus. Growth is usually slow, but sometimes the tumour becomes sufficiently large to interfere with mastication.

Treatment is excision of the tumour together with appropriate dental treatment. To prevent recurrence a small portion of alveolus at the base of the tumour should be removed.

*The Giant Cell (Myeloid) Epulis* is an osteoclastoma or 'benign giant cell tumour' (Chapter XIX) of the alveolus. It grows outwards and involves the gum, forming a soft maroon-coloured swelling (Fig. 58); ulceration and bleeding commonly follow.



FIG. 58. Myeloid epulis.

Until recently the orthodox treatment was to excise the superficial part of the tumour and scrape the remainder out of the bone. A more certain method, however, is to excise a block of tissue which includes the tumour and some surrounding healthy bone.

### **Carcinomata**

Carcinomata of the lip, tongue and mouth are all squamous-cell growths and will be considered first as a group; later we shall discuss them separately in more detail.

The patients are usually men over the age of 50. Chronic irritation and syphilis are predisposing causes. The irritation may be mechanical due to a jagged tooth or badly-made denture, or may be caused by excessive smoking or the consumption of raw spirits or highly seasoned foods.

The tumour may develop in the absence of any pre-existing lesion; it then begins as a small indurated nodule, and either breaks down to form a typical malignant ulcer with raised everted edge and indurated base or, less commonly, develops into a warty protuberance. Sometimes, however, especially on the tongue and lip, the tumour is preceded by a condition



known as *leukoplakia* (Fig. 59) in which irregular opaque whitish patches or raised plaques are formed as a result of thickening and keratinization of the epithelium. Deep fissures may develop between adjacent plaques and smooth glazed areas may be seen where plaques have been shed. Leukoplakia may remain unaltered for years, but malignant change is likely to occur sooner or later, often in the depths of one of the fissures. The first clinical indication of this change is usually pain or bleeding; if either of these symptoms appears a careful examination must be made, and will usually reveal an area of induration.



FIG. 59. Leukoplakia of the tongue.

Metastasis to cervical lymph nodes is common, but metastasis by the blood stream is very rare.

**Diagnosis.** In all patients with either leukoplakia or suspected carcinoma a Wassermann test should be done to determine whether syphilitic infection is present. In addition, when carcinoma is suspected, a biopsy should be performed to clinch the diagnosis.

**Principles of treatment.** In leukoplakia, smoking and the consumption of spirits are forbidden. Dental and anti-syphilitic treatment are given if required. A soothing ointment such as lanoline is prescribed for the lips, and if the tongue is affected an alkaline mouth wash is ordered. In some cases the lesions may be excised and the resulting defect repaired by a free skin graft or a flap. A careful watch is kept for the development of

malignant change and the patient is told to report *at once* if he experiences pain or bleeding.

In carcinoma the primary growth is treated either by radiotherapy or by excision, the choice depending on the site and extent of the lesion (*vide infra*) and also, to some extent, on the preference of the surgeon. Lymph node metastases are treated by block dissection if possible; if the disease is so advanced that this operation is impossible X-ray therapy is used as a palliative measure.

#### *Carcinoma of the lip*

Carcinoma of the lip is practically confined to men. It is common after the age of 50, but may occur from about 35 onwards. There is good evidence that in the day when men smoked clay pipes this habit was an etiological factor, and it is held by some that cigarette smoking is of importance to-day, though the evidence for this is less convincing.

The lesion is almost always on the lower lip, usually to one or other side of the mid-line, and presents the usual characteristics of a squamous-cell carcinoma (Fig. 60).



FIG. 60. Epithelioma of the lip.

Metastasis by the lymphatics seldom occurs for some months, or even a year, after the appearance of the primary growth. It occurs first to the submaxillary lymph nodes or, in the case of central growths, to the submental nodes; later the nodes of the jugular chain (Chapter XVIII) are involved.

*Diagnosis.* Every indurated lesion on the lip, especially on the lower lip, whether in the form of a nodule or an ulcer, should be regarded with suspicion. The diagnosis can usually be made clinically, but in cases of doubt a biopsy should be done. Confusion is occasionally caused by a gummatous ulcer and, when the lesion is on the upper lip, by a primary chancre; the Wassermann reaction, supplemented in the latter case by dark ground examination of the discharge for spirochaetes, however, should settle the matter.

*Treatment.* Small early lesions without evidence of lymph node involvement may be treated by X-ray therapy or by surgical excision of a V-shaped portion of lip containing the growth. The results in either case are excellent. The operation might be expected to cause severe deformity but in fact, if properly performed, results in a normal shaped mouth with only a faint linear scar at the line of suture of the skin.

With more extensive lesions surgery is mutilating and the primary growth should be treated by X-ray therapy, care being taken to avoid bone necrosis. Some surgeons do a block dissection of the nodes of the submental triangle and the submaxillary triangle on the side of the lesion about a fortnight after completing the treatment of the primary growth, even if there is no clinical evidence of lymph-node metastasis. If there is clinical evidence of metastasis a complete block dissection of the nodes of the neck on the affected side should be undertaken.

Patients with extensive lesions and inoperable metastases in the lymph nodes may be made more comfortable by a palliative course of X-ray therapy.

To sum up: treatment of epithelioma of the lip in its early stages is an extremely simple matter which causes little inconvenience to the patient and produces excellent results; neglected cases cause misery and ultimately death, and treatment is of little avail.

### *Carcinoma of the tongue*

Carcinoma of the tongue is one of the most dreadful diseases which afflicts mankind, yet if treated early the prospects of permanent cure are good. It is obvious, therefore, that early diagnosis is of paramount importance.

About 90 per cent of the sufferers are men, and most of them are over the age of 60. The importance of chronic irritation and syphilis as predisposing causes, and the significance of leukoplakia as a precancerous lesion, have already been discussed.

*Pathology and clinical features.* When carcinoma follows leukoplakia it may start in any of the numerous fissures and may pass unrecognized for a considerable time. In other cases it usually starts as a nodule on the edge of the buccal portion of the tongue; this gradually encroaches on the dorsum or the under surface of the tongue and at the same time breaks down to form a typical malignant ulcer (Fig. 61). Histologically the



FIG. 61. Carcinoma of the tongue.

degree of anaplasia varies widely in different tumours. Metastasis to the cervical lymph nodes occurs early. The submaxillary nodes and the nodes of the jugular chain are most often affected, but the submental nodes may also be involved if the growth is near the tip of the tongue. If the growth is anywhere on the posterior third of the tongue, or if it is on the anterior two-thirds and close to the mid-line, there may be metastasis to *contralateral nodes*; that is, to nodes on the right side of the neck when the primary lesion is on the left side of the tongue and vice versa.

In the early stages the patient may complain only of a feeling of stiffness in the tongue and a little discomfort after smoking or eating. Speech is unaffected, and on examination there may be only an indurated nodule or small ulcer. These features should suffice to suggest the diagnosis and a biopsy should be performed at once to confirm it.

If the lesion is untreated further symptoms appear, notably excessive salivation, foul breath, pain in the tongue or referred via the auriculo temporal nerve to the ear, and blurring of speech. On examination movement of the tongue is found to be restricted, the primary lesion covers a large area, and there may be enlarged and hard cervical nodes.

In the terminal stages salivation is extreme, and eating, swallowing and speaking become progressively more difficult and painful. The primary lesion may be hard to see owing to fixity of the tongue, but its size can be determined by palpation. There are usually fixed and stony hard lymph nodes in the neck.

Death comes as a merciful release. It may result from severe toxæmia due to secondary bacterial infection, from bronchopneumonia due to inhalation of septic material, or from secondary hæmorrhage due to erosion of a large vessel by the primary growth or a lymph node metastasis.

The differential diagnosis of malignant and non-malignant ulcers of the tongue will be discussed in Chapter XVI.

*Treatment.* In all cases appropriate dental treatment should be given and every effort made to clear up sepsis. Subsequent treatment depends on the stage of the disease.

1. *The early case without palpable nodes.* Growths in the anterior two-thirds of the tongue are usually treated by implantation of radium needles. Alternatively, part of the tongue, including the growth and a margin of healthy tissue, may be excised. If the tongue is carefully reconstructed the disability after this operation is much less than one might expect.

Growths in the posterior third of the tongue are not sufficiently accessible for implantation of needles and are treated by supervoltage X-ray therapy.

Block dissection of lymph nodes, though once advised as a routine procedure in every case of carcinoma of the tongue, is not performed nowadays unless there is clinical evidence of lymph node metastasis. The patient must be examined at monthly intervals, however, and block dissection must be undertaken without delay if nodes become enlarged.

2. *A more advanced but still operable case.* Again the primary growth is usually treated by radiotherapy, using needles for growths in the anterior two-thirds of the tongue and super-voltage X-ray therapy for those in the posterior third. The alternative is excision of the whole tongue after

splitting the *symphysis menti*, but this mutilating operation is rarely performed nowadays.

If there are lymph node metastases on one side of the neck a block dissection is performed 2 to 6 weeks after treatment of the primary growth. The nodes of the jugular chain down to the omohyoid muscle, the submaxillary and submental nodes, the surrounding fat, the sternomastoid muscle and the internal jugular vein are removed *en masse*. If there are bilateral lymph node metastases a block dissection may be performed first on one side and then, 6 weeks later, on the other. Removal of both internal jugular veins is permissible provided the operations are staged in this way. Alternatively, the nodes may be treated by deep X-ray therapy.

3. *The inoperable case.* Radium or X-ray therapy is used for the primary growth; X-ray therapy is also given to the nodes as a palliative measure.

### *Carcinoma of the mouth*

In this section we shall consider growths of the floor of the mouth, gums, cheek and palate.

A carcinoma may start in the floor of the mouth or may spread there from the tongue; in either case it usually takes the form of a typical malignant ulcer. Growths elsewhere in the mouth may present as malignant ulcers or as warty outgrowths.

Carcinomata of the mouth commonly metastasize to the cervical lymph nodes; they may also involve the mandible by direct spread. They may reach an advanced stage before pain and other symptoms cause the patient to seek medical advice.

*Treatment.* The primary growth is usually treated by X-ray therapy (Fig. 62). Care is necessary to avoid producing radio-necrosis of the jaw. Surgical excision is undertaken if the growth proves radio-resistant or recurs, or if it has spread to involve bone.

If the cervical lymph nodes are palpably enlarged, block dissection is performed as described in the section on carcinoma of the tongue.

## **Tumours of the Pharynx and Larynx**

### **Benign tumours**

The only benign tumour of importance in the regions under discussion is papilloma of the larynx. This occurs in both children and adults, and in the former may be multiple. It forms a cauliflower-like growth and is often situated on one of the vocal cords. Hoarseness is an early symptom; respiratory obstruction may occur in children, but is rare in adults. The



FIG. 62. Carcinoma of the alveolus.

A. Before treatment.

B. After X-ray therapy.  
(Mr. G. W. Blomfield's case)

diagnosis is made by laryngoscopy; if doubt exists about the pathological nature of the tumour a biopsy is performed.

Treatment is excision of the tumour through a laryngoscope. In children tracheostomy (that is, creation of an artificial opening into the trachea, through which the patient can breathe) is sometimes necessary to relieve respiratory obstruction; following this operation the tumour may disappear spontaneously.

### Carcinoma of the pharynx and larynx

In the faucial region and nasopharynx the usual malignant tumour is a highly anaplastic carcinoma which often contains much lymphoid tissue and is therefore sometimes called a *lympho-epithelioma*. The primary growth may be symptomless or may cause pain which is aggravated by swallowing; this pain may radiate to the jaw and ear. Metastasis to cervical lymph nodes usually occurs at an early stage. Most of these tumours are highly radio-sensitive and disappear for a time if treated by X-ray therapy. The prognosis is poor, however, because the tumour usually reappears within a few months and then resists all forms of treatment.

Tumours arising from the epiglottis, ary-epiglottic folds or pyriform fossae are usually typical squamous-cell carcinomata. They may invade the larynx and are then sometimes termed *extrinsic carcinomata of the larynx*.

Hoarseness and pain on swallowing are the main symptoms. X-ray therapy is the only practicable form of treatment but is curative in only a small proportion of cases.

Carcinomata of the post-cricoid portion of the pharynx, unlike carcinomata of the mouth and other parts of the pharynx, are much commoner in women than men. The main symptom is dysphagia (difficulty in swallowing). The growth is usually anaplastic and radio-sensitive, and is best treated by deep X-ray therapy. If it proves insensitive operative removal may be attempted. The operation is a formidable one but some permanent cures have been reported.

The term carcinoma of the larynx is commonly applied to two distinct types of lesion: intrinsic carcinoma arising in the larynx proper, and extrinsic carcinomata arising in the pharynx and spreading to the larynx (*vide supra*).

Intrinsic carcinoma of the larynx occurs much more commonly in men than in women. It usually takes the form of a squamous-cell carcinoma though basal-cell carcinomata and adenocarcinomata occur occasionally. The tumour may arise from a vocal cord or from the larynx below the cords (*sub-glottic carcinoma*). It usually forms a cauliflower-like mass. In the early stages the only symptom is hoarseness, but later dysphagia, abnormal salivation and cachexia appear. Metastasis to lymph nodes occurs much later than with carcinoma of the pharynx. The diagnosis is made by laryngoscopy combined with biopsy. In sub-glottic carcinoma the growth may not be visible, but its presence may be suggested by fixity of a vocal cord. In the early stages the tumour may be treated by radiotherapy or excised after opening the larynx by the operation of *laryngofissure*. In late cases the choice lies between radiotherapy and removal of the whole larynx. This operation is termed *laryngectomy*. It causes permanent loss of normal voice, but many patients can be trained by speech therapists to produce quite intelligible oesophageal speech.

### Tumours of the Oesophagus

Benign tumours of the oesophagus are extremely rare and of little practical importance.

Carcinoma of the oesophagus is usually of the squamous-cell variety, except at the lower end where adenocarcinoma and carcinoma simplex are common. The main symptom is dysphagia; the differential diagnosis of this symptom will be discussed in Chapter XXIII.

Until recently the treatment of carcinoma of the oesophagus was purely palliative. Nowadays, however, *partial oesophagectomy* offers some chance



of permanent cure. A portion of the oesophagus, extending from a point some distance above the tumour to the stomach, is removed and continuity of the alimentary tract is restored by anastomosing the stump of oesophagus to the stomach or to a loop of small gut pulled up into the mediastinum.

In inoperable cases supervoltage X-ray therapy may be of considerable palliative value. If the patient continues to have difficulty in swallowing even liquids a plastic tube is passed through the stricture and left permanently in place. This is preferable to the operation of *gastrostomy* (i.e. the creation of an artificial opening in the stomach for feeding purposes), which was once widely practised.

### **Tumours of the Salivary Glands**

Tumours of the salivary glands may be classified as follows:

- 1 Mixed salivary tumours.
- 2 Adenolymphomata.
- 3 Primary carcinomata.
- 4 Secondary carcinomata.

#### **Mixed salivary tumour**

Mixed salivary tumours are characterized histologically by the presence of epithelial cells, lymphocytes and a peculiar tissue which resembles either muroid connective tissue or cartilage. These tumours were once thought to be teratomata; it is now believed, however, that they are of epithelial origin and that the cartilage-like material is merely the connective tissue stroma of the tumour modified owing to the presence of mucin secreted by the epithelial cells. Mixed salivary tumours are benign and should be classed as adenomata, but they are very prone to recur after removal if even tiny fragments are left behind.

Mixed salivary tumours are common in the parotid but rare in the submaxillary salivary gland. Similar tumours sometimes develop elsewhere, notably, as we have already mentioned, from small mucous glands in the mouth, especially on the palate.

#### *Clinical features*

The patient is usually over the age of 30, but young adults and children are occasionally affected. The incidence is the same in both sexes.

The tumour grows slowly and has often been present for years when the patient is first seen. It is firm, smooth and painless, and usually shows some degree of mobility. If the body of the parotid is involved the swelling is situated between the mastoid process and the ascending ramus of the

mandible (Fig. 63); if the tumour is in the facial process of the parotid it lies over the masseter muscle a little below the zygomatic arch. If the submaxillary gland is involved the swelling is situated in the submaxillary triangle and can be palpated with one finger outside and one inside the mouth (Fig. 7). Facial paralysis never occurs with benign tumours except as a result of unskilful surgery. The cervical lymph nodes are not affected.



FIG. 63. Mixed parotid tumour.

### *Treatment*

A mixed tumour in the parotid should be excised together with some surrounding healthy parotid tissue, care being taken not to injure the facial nerve or its branches. In practice this often means excision of the superficial part of the gland. To minimize the risk of recurrence some surgeons insert radium needles into the cavity at the end of the operation and leave them in place for a week.

Mixed tumour of the submaxillary gland is treated by excision of the whole gland.

**Adenolymphoma**

This is a rare benign tumour which occurs in or near the lower pole of the parotid gland. Histologically it consists of small cystic spaces lined by pseudostratified columnar epithelium set in a stroma which is packed with lymphocytes, and has a well defined fibrous capsule. Clinically the lesion resembles a mixed parotid tumour.

*Treatment*

Simple enucleation is all that is required.

**Primary carcinoma**

Primary carcinoma is less common than mixed salivary tumour. As with the latter the parotid is affected much more often than the submaxillary gland.

The histological picture is very varied. The tumour may have the structure of a basal-cell carcinoma, an adeno-carcinoma or a carcinoma simplex. In many cases mucoid material is present similar to that seen in mixed tumours. Some carcinomata appear to develop in benign tumours, but the majority are probably malignant from the start.

The patient is usually over the age of 45; both sexes are equally affected. A salivary gland carcinoma grows more rapidly than a mixed tumour, but usually more slowly than carcinomata elsewhere in the body. In time it forms a stony hard fixed swelling, which infiltrates the gland and surrounding tissue, including in some cases the mandible, the skull or the pharynx. Metastasis may occur to cervical lymph nodes.

In carcinoma of the parotid pain is often severe; it may be localized to the tumour or felt in the distribution of the auriculo-temporal nerve. Facial paralysis commonly occurs.

*Treatment*

The affected gland should be excised completely and the patient given post-operative X-ray therapy. Injury to the facial nerve is inevitable, but is preferable to recurrence of the tumour following an inadequate operation. If the cervical lymph nodes are involved the operation is extended to include a block dissection.

**Secondary carcinoma**

Secondary carcinoma occurs occasionally especially in the submaxillary gland. It may arise in two ways:

1. Direct extension of a carcinoma in the vicinity. Carcinoma of the floor of the mouth, for instance, may spread to the submaxillary gland; carcinoma of the face to the parotid.

2. Spread from lymph node metastases; for example, from nodes in the submaxillary triangle to the submaxillary gland.

The treatment depends on the underlying condition.

### **The differential diagnosis of parotid tumours**

Parotid tumours must be distinguished from lymph node swellings and from enlargement of the parotid due to other causes.

Lymph node swellings (Chapter XVIII) can usually be recognized by their position; in addition, the presence of an inflammatory lesion or a tumour in the area drained by the nodes, or the enlargement of other groups of nodes, may provide a clue to the diagnosis.

The main causes of parotid enlargement apart from tumours are as follows:

1. Acute parotitis.
  - (1) Mumps.
  - (2) Acute suppurative parotitis.
2. Chronic parotitis.

Acute parotitis is easily distinguished by the short history. In mumps the swelling is nearly always bilateral.

Chronic parotitis presents a more difficult problem, especially if only one gland is affected. Usually, however, there is a history of recurring acute attacks and the swelling involves the whole gland. Facial paralysis does not occur.

The distinction between mixed salivary tumour and adenolymphoma on the one hand and carcinoma on the other has already been discussed.

### **Tumours of the Subcutaneous Tissue**

The only common tumour arising in the subcutaneous tissue is a lipoma; that is, a benign fatty tumour. A true lipoma is always encapsuled; diffuse masses of fat which occur as a result of endocrine disorders are not lipomata though the term is sometimes applied to them.

Subcutaneous lipomata may occur in any region, but are particularly common on the back of the neck. On the limbs or face they may be multiple and sometimes occur in symmetrical positions on the two sides of the body. They form lobulated well-defined swellings, attached to the skin (Fig. 64) but somewhat mobile in relation to deeper structures. They are soft in consistency and sometimes give the impression of fluctuation.



FIG. 64. Lipoma.

### *Treatment*

These tumours are quite harmless and never become malignant. They may be removed for cosmetic reasons if the patient so desires.

### **Tumours in Other Sites**

Tumours in various other sites including lymphoid tissue, bone and endocrine tissues will be discussed in later chapters.

## *Chapter XV*

### CYSTS

A cyst is a collection of fluid or semi-fluid material in a sac which has a lining membrane. Cysts are of many different kinds, but may be conveniently grouped under six headings:

1. Congenital cysts.
  - e.g. Sequestration dermoids.
  - Branchial cysts.
  - Thyroglossal cysts.
  - Congenital lymphatic cysts.
2. Retention cysts; that is, cysts due to obstruction of the duct of a gland.
  - e.g. Sebaceous cysts.
  - Mucous cysts.
3. Traumatic cysts.
  - (1) Post-traumatic epidermoid cysts.
  - (2) Encysted collections of blood or serum.
4. Parasitic cysts.
  - e.g. Hydatid cysts.
5. Neoplastic cysts.
  - (1) Epithelial tumours which are normally cystic; for example, ovarian teratomata.
  - (2) Cystic degeneration of a tumour.
6. Other types of cyst.

Most bone cysts and cysts of dental origin cannot be classified under any of the preceding headings, and are therefore included here.

#### **Clinical features and principles of treatment**

Most cysts are unilocular but some are multilocular. A unilocular cyst forms a smooth, rounded swelling, which may be mobile or fixed depending on the origin of the cyst and whether or not it is pedunculated. Fluctuation can be elicited unless the cyst is deeply situated or exceptionally tense, and transillumination can be demonstrated in special cases.

If the diagnosis cannot be made on clinical grounds it is often helpful to needle the cyst and aspirate some of the contents.

Treatment may be necessary for cosmetic reasons or because the cyst is causing pressure on neighbouring structures. If possible the whole cyst should be excised intact. If this cannot be done every bit of the lining membrane must be carefully removed or destroyed, otherwise recurrence is likely to occur.

Congenital cysts have been described in Chapter III, but some of the other varieties must now be considered in detail.

### **Sebaceous cyst**

A sebaceous cyst is a retention cyst caused by blockage of the duct of a sebaceous gland. Sebaceous cysts may occur anywhere on the surface of the body, but are particularly common on the scalp, behind the ears, and on the face, neck and scrotum. On the scalp (Fig. 65) they are often multiple and are sometimes called wens.

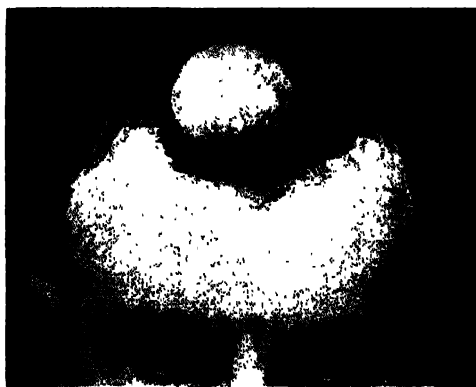


FIG. 65. Sebaceous cyst on the scalp.

A sebaceous cyst forms a tense, smooth, oval and painless swelling which is attached to the skin. The orifice of the duct can usually be seen and thick yellowish material—sebum—may be discharged through it from time to time. Fluctuation cannot normally be demonstrated unless the cyst becomes infected.

### *Complications*

*Infection.* This is a common complication. The cyst becomes softer and fluctuant, and the overlying skin slightly red, but there are usually no other signs of acute inflammation.

*Formation of a sebaceous horn.* This curious condition (Fig. 66) occurs occasionally when sebaceous material which escapes from the cyst is not wiped away but is allowed to dry on the surface. As more material is



FIG. 66. A sebaceous horn.

discharged the 'horn' increases in length, sometimes up to an inch or more.

*Ulceration.* Occasionally an infected cyst ulcerates through the overlying skin. Such a lesion on the scalp is sometimes known as 'Cock's peculiar tumour' and has been mistaken for an epithelioma.

*Malignant change.* This has been reported, but is extremely rare.

### *Treatment*

Uncomplicated cysts should be removed under local anaesthesia. The whole cyst may be excised together with a small ellipse of skin; alternatively the cyst may be split in its long axis, the contents squeezed out and the lining membrane removed in two halves. The whole of the lining must be removed or the cyst will recur.

Infected cysts should be incised under gas and oxygen or thiopentone anaesthesia and the lining curetted out as completely as possible. If, as is often the case, this is followed by recurrence the cyst should be dissected out before further infection occurs.

### **Mucous cyst—ranula**

Small mucous retention cysts are common in the mouth and on the mucosal surface of the lips. They form small rounded translucent painless swellings which are easily recognized. Sometimes they burst spontaneously and then re-form.



A large mucous cyst sometimes occurs in the floor of the mouth slightly to one side of the mid-line, and is known as a *ranula* (Fig. 67). The tongue may be displaced upwards or to one side. A very large ranula occasionally extends downwards between the mylohyoid muscles and appears in the submental triangle of the neck.



FIG. 67. Ranula.

### *Treatment*

Small mucous cysts should be excised under local anaesthesia. A ranula presents a more difficult problem. Ideally it should be excised entire, but in practice the thin wall usually bursts during removal. If this happens it is best to remove as much of the wall as possible and destroy the remainder with diathermy. Alternatively, the 'roof' of the cyst may be removed and the mucosa of the remainder sutured to that of the mouth, so that cyst and mouth form a continuous cavity.

### **Post-traumatic epidermoid cysts**

A post-traumatic epidermoid is a cyst lined by squamous epithelium which sometimes appears on the hand or foot following injury. These cysts were originally called implantation dermoids because it was believed that they arose from surface epithelium displaced into the deeper tissues by a punctured or incised wound. They may, however, follow bruising without any external wound and it seems probable that they arise from sweat gland epithelium.

There is usually an interval of several months between the injury and the appearance of the cyst. Common sites are the flexor surface of a finger, the palm of the hand and the sole of the foot. The cyst is usually small, rounded and tense, and is situated immediately beneath the skin. The diagnosis should present no difficulty to anyone aware of the condition.

The treatment is excision.

### Cysts and tumours of dental origin

Cysts and solid tumours which arise in connection with the teeth are often referred to generically as *odontomes*. The word 'tumour' is not used in its strict sense because, with one possible exception, none of the members of the group is a true neoplasm.

Odontomes are discussed at length in textbooks of dental surgery and only a brief account of the more important types will be given here.

#### 1. Dental cyst

A dental cyst occurs in relation to the root of a dead or carious tooth, and is generally regarded as being of inflammatory origin. It may occur anywhere in either the upper or lower jaw.



FIG. 68. Radiograph of a large dental cyst in the lower jaw.

The cyst may be lined with squamous epithelium, granulation tissue or a mixture of both. It is characteristically unilocular, and presents a sharp clear outline in a radiograph (Fig. 68).

*Treatment.* The cyst is exposed by incising the muco-periosteum and removing the bony wall on its labial aspect. The further steps in the operation vary according to circumstances. It is sometimes possible to enucleate the entire cyst lining and when this is done the incision is closed. Failing this, as much of the lining as possible is removed and the cavity is widely saucerized, after which a muco-periosteal flap is turned into the cavity and held in place with a pack; or alternatively the cyst is *marsupialized*, i.e. the cavity is widely saucerized and the cyst lining, which is not removed, is sutured to the muco-periosteum.

## 2. *Dentigerous cyst or 'follicular odontome'*

A dentigerous cyst arises in connection with an unerupted tooth, most commonly in the molar region of the mandible.

The etiology is uncertain, but it has been suggested that caries of a deciduous tooth leads to the formation of a dental cyst which then envelops a developing permanent tooth and prevents its eruption.



FIG. 69. Radiograph of a dentigerous cyst in a child. Compare the appearance of the cyst with that of the unerupted *normal* tooth.

A dentigerous cyst may lead to considerable expansion of the jaw and may cause aching pain.

The diagnosis is suggested by the occurrence of a cyst associated with the absence of a tooth; it is confirmed by X-ray, which reveals an unerupted tooth with its crown lying within the cavity of the cyst or embedded in the cyst wall (Fig. 69).

*Treatment.* If the unerupted tooth is in good position and is not a supernumerary tooth the cyst is opened widely into the mouth and a pack is placed round the tooth in the hope that it will erupt normally. If on the other hand the tooth is in poor position or is supernumerary it is removed together with the cyst lining.

### 3. *Adamantinoma*

Adamantinoma is more common in women than men. It occurs almost exclusively in the lower jaw.

Again the etiology is uncertain, but the condition is believed to be a true neoplasm. Some authorities hold that it is of the same nature as basal-cell carcinoma of the skin.

Growth is slow, but over a period of years the jaw becomes expanded. The bone is replaced by masses of epithelial cells and cyst formation usually, though not invariably, occurs. The cysts are lined with either squamous or columnar epithelium and are separated from each other by fibrous or bony septa (Fig. 70). Though adamantinomata are locally



FIG. 70. Radiograph showing multilocular cystic disease of the jaw due to adamantinoma.

invasive they do not usually metastasize. A few cases of frankly malignant adamantinomata with distant metastases have, however, been reported.

Removal of the tumour from within the mouth is usually impracticable, and an operation from the external aspect is therefore necessary. When

the growth is large this takes the form of partial resection of the mandible and repair of the defect with a bone graft. The graft is usually taken from the crest of the ilium.

#### *4. Solid odontomes*

Various solid odontomes have been described, including composite odontomes derived from irregular growth of one or more whole tooth germs, myxomatous and fibrous odontomes, dentinomes and cementomes. All of these are relatively rare in man, though some are common in herbivorous animals. When they occur removal from the mouth is often possible; if this is not the case treatment follows the lines described for multilocular cystic disease.

### **Bone cysts**

Bone cysts are described in Chapter XIX.

## Chapter XVI

# ULCERATION, GANGRENE, SINUSES AND FISTULAE

An ulcer is a breach of continuity of a surface resulting from death of superficial tissue.

### Examination of an ulcer

Every ulcer has an edge, a floor and a base. The edge and floor are examined by inspection. The base is the zone of tissue immediately underlying the floor; it is examined by palpation.

*The edge.* Note the shape of the ulcer—round, oval, serpiginous or irregular—and its size. Observe whether the edge is clean cut, undermined, shelving or everted (Fig. 71).

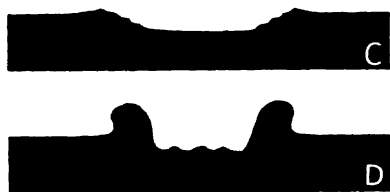


FIG. 71. Ulcers in profile.

- A. Clean-cut edge characteristic of gummatous ulcers.
- B. Undermined edge characteristic of tuberculous ulcers.
- C. Shelving edge characteristic of many simple ulcers.
- D. Everted edge characteristic of carcinomatous ulcers.

*The floor.* The floor may consist of granulation tissue, fibrous tissue, slough or new growth. Note its appearance and the character of any discharge.

*The base.* Determine by palpation the degree of induration and the extent of fixation to deeper structures.

**Types of ulcer**

Three main types of ulcer are recognized:

1. Simple ulcers.
2. Specific ulcers.
3. Neoplastic ulcers.

**Simple ulcers**

Simple ulcers may be acute or chronic. They are due to a variety of causes, including trauma, vascular causes (for example, varicose ulcer and post-thrombotic ulcer—Chapter XVII), nutritional causes, lesions of the nervous system ('trophic ulcers') and non-specific infection.

Three stages may be recognized during the clinical course of a simple ulcer:

1. *The stage of active extension.* The edge shows evidence of acute inflammation, the base is oedematous and indurated, the floor is covered with a yellowish slough, and there may be a discharge of pus.

2. *The transitional stage.* The discharge is diminished, sloughs are becoming separated and replaced by granulation tissue, and the base is less indurated. Some chronic ulcers may remain in this stage for months or even years; an acute ulcer, on the other hand, may progress to the next stage within a day or two.

3. *The stage of healing.* The edge becomes shelving and epithelium begins to grow in and repair the defect. Granulation tissue gradually gives place to firm fibrous tissue.

*Treatment of simple ulcers.* Whenever possible the cause of the ulcer should be determined and treated appropriately.

Further measures depend on the site of the ulcer. If it is situated on a limb a sterile dressing is applied and the limb is elevated or provided with an elastic support. Various antiseptics are sometimes applied. A proprietary preparation known as *Furacin* is very useful in controlling infection and *eusol* promotes the separation of sloughs. In chronic cases skin grafting may be required. Usually the graft is applied to the floor of the ulcer after the infection has subsided; occasionally, however, the whole ulcer is excised with a small margin of healthy tissue and the graft is applied to the raw area thus produced.

The treatment of simple ulcers of the lip and tongue is discussed below.

**Specific ulcers**

A specific ulcer is an ulcer caused by one of the specific infectious diseases such as tuberculosis, syphilis or Vincent's angina. The characteristics of

the more important specific ulcers have already been described (Chapters XII and XIII); the treatment is that of the underlying disease.

### **Neoplastic ulcers**

Neoplastic ulcers are usually carcinomatous. The underlying lesion may be a basal-celled carcinoma, a squamous-celled carcinoma or an adenocarcinoma arising on an epithelial surface. The characteristics and treatment have been discussed in Chapter XIV.

In addition, a malignant tumour arising subjacent to an epithelial surface, whether carcinoma or sarcoma, may spread directly to involve the surface and so lead to ulceration. This condition must be distinguished from simple ulceration due to the pressure of an expanding benign neoplasm in the subcutaneous tissue or submucosa.

### **Ulcers of the lips**

*Simple ulcers.* Simple ulcers commonly occur as a result of exposure to wind or sunshine. The lower lip is most affected, and the ulcers take the form of painful cracks. The treatment is to prevent further exposure and to apply a bland ointment such as zinc cream. Simple ulcers may also result from vitamin deficiency; cracks at the angles of the mouth, for example, are characteristic of riboflavin deficiency.

*Specific ulcers.* Specific ulcers of the lips are of two main kinds; herpetic and syphilitic.

Herpetic ulcers occur in the disease known as *herpes simplex*. This is due to infection by a filtrable virus akin to, but distinct from, the virus responsible for the more serious condition of *herpes zoster* (Chapter XXI). Small submucous vesicles appear on one or both lips, commonly when the patient is suffering from an acute respiratory infection. The vesicles break down to form superficial ulcers which usually heal rapidly. Recent investigations indicate that the patient usually becomes infected with the herpes virus in childhood, and continues to harbour it for the rest of his life; for most of the time, however, there are no symptoms and the existence of the infection can be demonstrated only by immunological tests.

Syphilitic ulcers may occur in both the acquired and congenital forms of the disease. In acquired syphilis the ulcer may take the form of a chancre (primary syphilis), a snail-track ulcer (secondary syphilis) or a gumma (tertiary syphilis).

A primary chancre of the lip may occur by direct infection from a patient with primary or secondary syphilis, or indirectly through using a contaminated drinking vessel. The upper lip is affected more often than the lower, and the skin is usually involved more extensively than the



mucous membrane. The lesion resembles a genital chancre but is usually not so indurated. It begins as a raised nodule, but soon breaks down to form a painless ulcer having a sloping edge and a floor of granulation tissue. There is a thin watery discharge containing numerous spirochaetes. The submaxillary lymph nodes are usually markedly enlarged. The ulcer may heal spontaneously in a few weeks. The diagnosis is confirmed by microscopic examination of the discharge for spirochaetes using dark ground illumination, and by the Wassermann or Kahn test.

In secondary syphilis mucous patches and snail-track ulcers may occur on the inner aspects of both lips. They have been described in Chapter XIII.

A gummatous ulcer occurs sometimes on the lower lip and very occasionally on the upper. It has a circular or serpiginous outline, a clean-cut edge giving a 'punched-out' appearance, and a 'wash-leather' slough forming the floor. The related lymph nodes are usually not enlarged. The Wassermann and Kahn tests are positive.

Painless fissures may develop at the angles of the mouth in congenital syphilis. This condition is known as rhagades. Other stigmata of the disease are usually present (Chapter XIII).

*Neoplastic ulcers.* Two types of neoplastic ulcer occur on the lips; one is due to squamous-cell carcinoma, the other (rodent ulcer) to basal-cell carcinoma.

The squamous-cell types is common. It occurs usually on the lower lip and has the characteristic raised everted edge, irregular floor and indurated base already described (Chapter XIV). Rodent ulcer is rare and when present is usually on the upper lip.

The clinical features of the various ulcers which occur on the lips are summarized in Table IV. The most important differential diagnosis is between squamous-cell carcinoma and gumma. This can usually be made on the clinical findings and the result of the Wassermann reaction, but in doubtful cases a biopsy should be performed.

### **Ulcers of the tongue**

*Simple ulcers.* Simple ulcers of the tongue are of two main types, traumatic dental ulcers and dyspeptic ulcers.

Traumatic dental ulcers are caused by jagged teeth or, occasionally, by faulty dentures. They usually occur on the side of the tongue and are painful and tender. There is a surrounding zone of hyperaemia. The edges are oedematous; sometimes so oedematous that the floor of the ulcer is hidden from view. If the cause is removed the ulcer usually heals rapidly;

TABLE IV. Types of ulcer occurring on the lips

Etiology and type of ulcer	Usual site	Clinical features	Special tests used to confirm diagnosis
Simple ulcer due to exposure	Both lips, but especially lower	Painful cracks	
Simple ulcer due to riboflavin deficiency	Angles of mouth	Relatively painless cracks	Therapeutic test with riboflavin
Herpes simplex	Both lips, but especially upper	Small vesicles which ulcerate and then heal spontaneously. Appear following exposure to cold or in association with respiratory infections	
Primary syphilis—chancres	Upper lip	Painless ulcer with sloping edge and granulating floor. Thin watery discharge. Submaxillary lymph nodes enlarged. Ulcer may heal spontaneously in a few weeks	(1) Microscopic examination of discharge using dark ground illumination (2) Wassermann reaction or Kahn test
Secondary syphilis—mucous	Mucosal surface of lips	Superficial 'snail-track' ulcer covered with mucoid exudate. Often painful. Other evidence of secondary syphilis present	Wassermann reaction or Kahn test
Tertiary syphilis—gummatous ulcer	Lower lip	Painless circular or serpigenous ulcer with punched-out edge and sloughing floor.	Wassermann reaction or Kahn test
Congenital syphilis—rhagades	Angles of mouth	Painless fissures. Other evidence of congenital syphilis	Wassermann reaction (mother and child) or Kahn test
Epithelioma	Lower lip	Painless ulcer with raised everted edge, irregular floor and indurated base. Regional nodes involved late in disease	Biopsy
Rodent ulcer	Upper lip	Painless superficial ulcer with 'beaded' edge. Regional nodes never involved	Biopsy

if, on the other hand, the cause is not removed the tissue around the ulcer become indurated and eventually malignant change may occur.

Dyspeptic ulcers—so-called because they are sometimes associated with attacks of dyspepsia (indigestion)—commonly occur on the tip or dorsum of the tongue and on the inner aspect of the cheeks. The etiology is obscure, but some people seem to be particularly prone to develop them. They are small and circular with a sloping oedematous edge and a yellowish floor. Surrounding the ulcer is a well-marked zone of hyperaemia. Dyspeptic ulcers are decidedly painful, but they heal spontaneously within a few days and no treatment is required.

*Specific ulcers.* There are two important specific ulcers of the tongue: tuberculous ulcer and gummatous ulcer.

Tuberculous ulcers occur only in patients with laryngeal or advanced pulmonary tuberculosis. The ulcer is usually situated near the tip of the tongue, but may occur anywhere. It may take the form of a narrow fissure or a shallow ulcer with undermined edges and unhealthy granulations in the floor. Pain is a characteristic symptom and the ulcer is tender on examination. The submental or submaxillary lymph nodes may be enlarged. Treatment is directed to the underlying condition.

Gummatous ulcer occurs on the dorsum of the tongue, and there is often associated leukoplakia. The ulcer is painless and has the usual punched-out appearance and sloughing floor. The Wassermann reaction (and Kahn test) are positive, but biopsy is sometimes necessary to confirm the diagnosis and exclude the possibility of carcinoma.

*Neoplastic ulcers.* Neoplastic ulcer of the tongue is always due to squamous-celled carcinoma. The ulcer is initially painless, but later may be very painful. It has the usual raised everted edge and indurated base. Foul breath and salivation are common symptoms and movement of the tongue is restricted in late cases. The regional lymph nodes are often enlarged. The diagnosis is confirmed by biopsy. Treatment has been discussed in Chapter XIV.

The characteristic features of the various ulcers which occur on the tongue are summarized in Table V. It is a sound rule to regard every chronic ulcer of the tongue as probably carcinomatous until the contrary has been proved, and in all doubtful cases a biopsy must be performed. A positive Wassermann test proves that the patient has syphilis, but *it does not exclude cancer*, since both conditions may be present.

### **Ulcers of the stomach and duodenum**

A special type of simple ulcer known as *peptic ulcer* occurs in the stomach and duodenum. The only other type of ulcer which occurs in this region is

TABLE V. Lingual ulcers

Etiology and type of ulcer	Usual site	Clinical features	Special tests used to confirm diagnosis
Traumatic dental ulcer	Edge	Painful; moderately deep ulcer with sloping edges and surrounding hyperaemia. Cause usually apparent and ulcer heals when this is removed	
'Dyspeptic'	Tip or dorsum	Painful; small circular ulcer with oedematous edge, yellow floor and surrounding hyperaemia. Heals spontaneously in a few days	
Tuberculous	Most commonly tip, but may occur anywhere	Painful; shallow ulcer with undermined edge and unhealthy granulations in floor. Associated laryngeal or advanced pulmonary T.B. Regional lymph nodes often enlarged	Sputum for tubercle bacilli
Gummatous	Dorsum	Painless; punched-out ulcer with sloughing floor. Often areas of leukoplakia elsewhere on tongue	Wassermann reaction Biopsy
Epitheliomatous	Edge	Initially painless but later very painful. Heaped-up everted edge and indurated base. Movements of tongue restricted in later stages. Foul breath and salivation. Regional nodes enlarged except in early cases	Biopsy

*carcinomatous ulcer*; this is common in the stomach, but never occurs in the duodenum.

### *Peptic ulcer*

*Etiology.* Peptic ulcer may occur at any age, but is rare in children and most commonly develops in early adult or middle life. Duodenal ulcer is more common than gastric ulcer. It occurs much more frequently in men, whereas gastric ulcer is about equally common in the two sexes. Some people appear to have an inherited predisposition to ulcer. Worry and irregular meals are definite etiological factors, and dental sepsis and excessive smoking may play a part in some cases.

The mechanism by which the ulcer is produced is not fully understood. The gastric juice—which normally contains hydrochloric acid and a proteolytic enzyme known as *pepsin*—plays an important role, since peptic ulcers only develop in the presence of this juice, and are especially common when the acidity is unusually high. Normally the mucous membrane is able to withstand the action of the juice; if this were not so we would all have ulcers. It appears, therefore, that for ulceration to occur a localized area of mucosa must be devitalized and so rendered incapable of resisting digestion. Despite much research the cause of this devitalization is unknown.

*Symptoms and signs.* Peptic ulcers are of two kinds: acute and chronic.

Acute ulcers either heal rapidly or develop into chronic ulcers. They may bleed or occasionally perforate (*vide infra*), but apart from this usually pass unrecognized.

Chronic peptic ulcer causes periodic attacks of upper abdominal pain bearing a definite time-relationship to meals. With gastric ulcers the pain usually comes on  $\frac{1}{2}$  to 2 hours after a meal; with duodenal ulcers it starts  $2\frac{1}{2}$  to  $3\frac{1}{2}$  hours after a meal and is relieved by food. The attacks usually last for several weeks; they alternate with remissions lasting weeks or months during which the patient is free from symptoms.

On examination of the abdomen during an attack there is usually a tender spot in the epigastrium, but often nothing else abnormal. The diagnosis is suggested by the history and is confirmed by radiological examination after giving the patient a barium meal. Valuable information may also be obtained by measuring the amount of acid produced in response to an injection of histamine. An antihistamine drug which inhibits most of the effects of histamine but not its capacity to stimulate acid secretion, is given prior to the histamine injection.

*Complications.* The most important complications are *perforation* of the ulcer into the general peritoneal cavity, narrowing of the pylorus (*pyloric stenosis*) and *haemorrhage*. Following haemorrhage the patient may vomit dark material resembling coffee grounds; this is termed *haematemesis*. Alternatively, the blood may pass through the intestines, causing the stools to become black and tarry; this is known as *melaena*.

*Treatment.* An uncomplicated ulcer is usually treated medically in the first instance; if this fails surgery may be required. Two types of operation are in current use for the treatment of chronic duodenal ulcer: (a) division of the vagus nerves (*vagotomy*) in the abdomen combined with *gastro-jejunostomy* or *pyloroplasty* (an operation to increase the diameter of the pylorus), or (b) *partial gastrectomy*. The secretion of acid is stimulated partly by impulses transmitted in the vagus nerves and partly by a hormone

(*gastrin*) which is secreted in the distal part of the stomach. Vagotomy removes the nervous stimulus and so reduces acid secretion; it also slows emptying of the stomach, but this is corrected by the gastrojejunostomy or pyloroplasty. In partial gastrectomy about two-thirds of the stomach is removed together with a small portion of duodenum; the duodenal stump is closed and continuity of the alimentary canal is restored by anastomosing the remnant of stomach to the jejunum. This removes the area of the stomach in which gastrin is formed, and also part of the area in which acid secretion occurs.

The usual operation for chronic gastric ulcer is partial gastrectomy, but in this condition the remnant of stomach is sometimes anastomosed to the duodenum instead of to the jejunum.

Operative treatment may also be required for the treatment of perforation and pyloric stenosis, and sometimes for severe haemorrhage if it fails to respond rapidly to medical treatment.

### Gangrene

Gangrene is defined as the death of tissue *en masse*. Two main types are described: dry gangrene and moist gangrene.

In dry gangrene or 'mummification' the affected part is shrivelled, hard, dry and dark brown or black in colour because of the presence of altered blood. The dead tissue gradually becomes separated from the living by a zone of granulation tissue. The junction of dead and living tissue is called the 'line of demarcation' (Fig. 72).

In moist gangrene there may be no definite line of demarcation. The gangrenous part is swollen, black or green in colour and often shows large



FIG. 72. Dry gangrene.

blisters containing foul-smelling fluid; these changes arise from infection and putrefaction.

### **Causes of gangrene**

The causes of gangrene may be classified as follows:

#### *1. Mechanical injury*

Severe crushing sometimes results in gangrene. This is usually termed 'direct traumatic gangrene'.

#### *2. Thermal and chemical injury.*

Thermal injury may take the form of either burns or frostbite. The lesions caused by corrosive poisons provide an example of gangrene due to chemical injury.

#### *3. Diminution of the arterial blood supply to the part.*

This may result from disease which causes narrowing of the arteries, or from arterial injuries. Gangrene due to arterial disease is common, especially in elderly people. Gangrene due to arterial injury is sometimes known as 'indirect traumatic gangrene'.

#### *4. Infection*

(1) Acute pyogenic infection. The slough in a boil or carbuncle is an example of gangrene due to this cause.

(2) Gas gangrene. This has been described in Chapter XII.

#### *5. Diabetes*

Diabetes is a very common cause of gangrene, and the urine should therefore be tested for sugar in every patient.

Diabetes leads to gangrene partly because it is often associated with severe arterial disease, and partly because the resistance of the tissues to infection is reduced when the blood sugar is abnormally high.

### **Clinical course and treatment**

Very small areas of gangrene may be absorbed by the action of phagocytic cells. Larger areas become separated from the living tissue by a layer of granulation tissue and are cast off. In diabetics moist gangrene is common, and infection may spread from the dead to the living tissue with serious consequences.

In planning treatment it is important to know the cause of the gangrene. Disorders of arterial blood supply may be indicated by coldness or blueness

of the part adjacent to the gangrenous area or by absence of arterial pulsation. Diabetes is diagnosed by demonstrating glycosuria and an abnormal blood sugar curve.

In cases of dry gangrene the affected part is kept dry and aseptic. Until recently it was usual to keep the part warm in the hope that this would result in an increased blood supply. It is now realized that the rise in temperature, by increasing the metabolic activity of tissue not actually dead, causes a greater demand for blood than can be supplied by the vasodilatation it brings about. The present practice, therefore, is to keep the affected part cool, but to try to provide general vasodilatation by heating the rest of the patient's body.

Sometimes the blood supply to a limb may be increased by interrupting its sympathetic nerve supply. This operation is termed *sympathectomy*. It may be undertaken to prevent the development of gangrene or in the hope of limiting the extent of the process.

When a line of demarcation appears spontaneous separation of the gangrenous part may be awaited, but if this is unduly delayed local amputation is undertaken.

In cases of moist spreading gangrene early amputation may be necessary to save the patient's life. Because arterial disease is usually widespread in these cases the amputation often has to be performed at a high level; for example, through the lower third of the thigh for gangrene of the foot. Antibiotics are given in large doses before and after operation.

## **Sinuses and Fistulae**

### **Sinus**

A sinus is a blind track opening on the surface of the body, and lined by granulation tissue, epithelium or a mixture of the two.

A few sinuses are congenital, but the majority are acquired as a result of chronic inflammation. This often takes the form of chronic suppuration round foreign or dead material (Fig. 73), such as a piece of shrapnel, an unabsorbed catgut stitch, a sequestrum or the root of a dead tooth; but sinuses also result from certain specific infections, notably tuberculosis and actinomycosis.

### *Treatment*

Congenital sinuses should be excised completely.

With acquired sinuses it is essential to find and remove the underlying cause. This is usually all that is required, but if the track has become





FIG. 73. Discharging sinus in a patient with osteomyelitis. The sinus leads down to a sequestrum (Chapter XIX).

epithelialized, or if its wall is rigid from chronic fibrosis, it must be excised before healing will take place.

### **Fistula**

A fistula is an abnormal communication between two epithelial-lined viscera or between one such viscus and the surface of the body.

Fistulae may be congenital or acquired, and the acquired type may be traumatic, inflammatory or neoplastic in origin. Traumatic fistulae may occur accidentally or may be produced deliberately by surgical operations (Chapter II).

An acquired fistula which is not associated with obstruction to a natural passage may close spontaneously, but fistulae associated with partial or complete obstruction remain open until this has been removed. A fistula made into the bladder to relieve urinary obstruction caused by enlargement of the prostate gland, for instance, will remain open indefinitely as long

as the obstruction remains, but once the prostate has been removed it usually closes in a few weeks.

### *Principles of treatment*

The treatment of certain congenital fistulae has been discussed in Chapter III.

The treatment of an acquired fistula depends on the cause. If there is associated obstruction to a natural passage the obstruction must be removed or, if this is impossible, bypassed by a short-circuiting operation. The fistula may then heal spontaneously; if this does not happen the fistula is excised and the visceral connections are closed. Another procedure, specially applicable to a fistula between the skin and the duct or secreting acini of a gland, is to abolish the secretion by denervating the gland or by destroying it by irradiation.

The application of some of these principles to parotid fistulae is illustrated below.

### **Parotid fistulae**

Parotid fistulae are of two types: fistulae of the gland proper and fistulae of Stenson's duct. In both types there is a discharge of saliva on to the face, which is increased by taking food.

#### *Fistulae of the gland proper*

A fistula of the gland proper may be caused by a wound, or may develop as a complication of suppurative parotitis when an abscess connected with secreting tissue is incised or ruptures through the skin. Spontaneous healing usually occurs, but if the fistula persists the secretion of the gland may be abolished as described below.

#### *Fistulae of Stenson's duct*

Fistulae of Stenson's duct are invariably due to wounds. The development of a fistula following injury can usually be prevented by immediate repair, but unfortunately the damage to the duct often passes unrecognized at the time. If the duct is only partly divided spontaneous healing occurs; if, however, it is completely divided the distal (buccal) end of the duct becomes shrunken, a persistent fistula results and repair is impossible.

### *Treatment*

If the fistula arises from the portion of the duct which lies anterior to the masseter muscle, treatment is easy; all that is required is to make a new

opening between the proximal portion of the duct and the mouth by a simple plastic operation. If, on the other hand, the fistula arises further back in the duct plastic operations are difficult and often unsuccessful, and the best treatment is to abolish the secretion of the gland by avulsing the auriculo-temporal nerve or destroying the secretory epithelium with X-rays.

## *Chapter XVII*

# DISORDERS OF BLOOD VESSELS

### Veins

#### Injuries

*Wounds of veins* cause haemorrhage (Chapter VIII). A further danger with an extensive wound of a large vein is that air may enter the circulation in sufficient quantity to cause frothing in the chambers of the heart and seriously interfere with the heart's action. This is known as *air embolism*.

*Rupture* in the absence of a penetrating wound rarely occurs with normal veins, but is a common complication of varicose veins (*vide infra*). The haemorrhage may appear alarming, but is easily controlled by elevating the limb or applying a firm pad.

*Contusion* of a vein may result in thrombosis (*vide infra*).

#### Thrombosis

Thrombosis means intravascular clotting; it may occur in both arteries and veins but is particularly common in the latter. Thrombosis associated with inflammation in the wall of a vein is called *thrombophlebitis*.

The predisposing causes of thrombosis are:

1. Injury or disease of the vessel wall.
2. Slowing of the rate of blood flow through the vessel.
3. Increased coagulability of the blood.

#### *Post-operative thrombosis*

Venous thrombosis is particularly common after operations. It usually occurs in the deep veins of the calf, whence it may spread to the femoral and iliac veins, or in the veins of the pelvis. The cause of post-operative thrombosis is still imperfectly understood, but slowing of the blood stream, and dehydration leading to increased coagulability of the blood, are among the factors responsible.

*Clinical features.* If the veins of the calf are affected the leg becomes swollen, though this may only be apparent if its circumference is measured and compared with that of the opposite limb; the muscles become tender, and pain is felt in the calf when the foot is passively dorsiflexed. If the process spreads to the femoral and iliac veins the whole limb may become

bluish in colour and grossly oedematous, and there is pain and tenderness along the line of the femoral vein.

A slight elevation of temperature usually occurs, especially in the evening.

The condition is dangerous because it may lead to pulmonary embolism (Chapter II).

A more remote complication which may develop months or years after femoral vein thrombosis is the development of chronic ulcer of the leg (*post-thrombotic ulcer*) similar in appearance to a varicose ulcer (*vide infra*). This is liable to develop when, as often happens, the thrombosed vein becomes recanalated, because the valves in the deep veins of the limb, and also in the communicating veins which join the superficial and deep veins, are left incompetent, and this leads to impairment of the circulation and anoxia of the tissues.

*Treatment.* The patient is kept in bed with the affected limb elevated. Anticoagulant drugs are administered to limit spread of the clot. Provided no contraindication exists heparin, which acts quickly, is given intravenously, and phenindione by mouth. As soon as the phenindione has begun to take effect, as shown by an increase in the prothrombin time, administration of heparin is stopped. Anticoagulant drugs occasionally cause serious haemorrhage; they should therefore only be used under strict medical supervision.

In femoral thrombosis the above measures are sometimes not sufficient and it may be necessary to remove the thrombus and ligate the vein at a higher level to overcome the danger of embolism.

### **Varicose veins**

Varicose veins are veins which have become *dilated, tortuous, elongated* and *incompetent* (that is, incapable of returning blood to the heart against gravity). They rarely occur before the age of 20, but become common thereafter, especially in women. The superficial veins of the legs are most commonly affected (Fig. 74).

In an early case, apart from visible change in the veins, there are no symptoms other than slight aching in the legs after standing for a long time. Later, continued venous stasis leads to impaired nutrition of the skin and subcutaneous tissues, and ulceration (Fig. 75) commonly ensues.

Other complications which may occur are thrombophlebitis and rupture. *Thrombophlebitis* is often precipitated by a minor injury. The affected segment of vein becomes hard, swollen and tender, and there is slight reddening and oedema of the overlying skin. There may be mild fever.

*Rupture* may be caused by injury or may occur spontaneously. Bleeding may be profuse but stops as soon as the limb is elevated.

### *Treatment*

In early cases a sclerosing fluid is injected to produce thrombosis of the veins. In advanced cases, when there is incompetence of the valves of the great saphenous vein throughout its length, this vein and its tributaries are ligated in the groin. Sometimes it is also necessary to strip out the whole long saphenous vein, or to ligate or excise dilated veins below the knee.

For a varicose ulcer elastoplast is applied to the leg and the patient is made to rest as far as possible. Vein ligation is required to prevent recurrence of the ulcer.



FIG. 74. Varicose veins.



FIG. 75. Varicose ulcer.

## **Haemorrhoids**

Haemorrhoids are due to varicosity of veins around the anus and in the anal canal. Two main varieties are described: external haemorrhoids and internal haemorrhoids.

*External haemorrhoids* are situated entirely outside the anus. They cause no symptoms unless thrombosis occurs when a painful tense purple swelling develops at the anal margin. The treatment of a thrombosed

external haemorrhoid is to make a small incision under local anaesthesia and evacuate the clot.

*Internal haemorrhoids* develop inside the anus. There are usually three haemorrhoids, each consisting of dilated veins and connective tissue covered by mucous membrane, and containing a branch of the superior haemorrhoidal artery. The main symptoms are bleeding and prolapse; pain does not occur in the absence of complications. The diagnosis is confirmed by examination of the anal canal through a proctoscope (Chapter I).

The complications include thrombosis, ulceration and strangulation. In strangulation the haemorrhoids are prolapsed and so tightly gripped by the anal sphincter that their blood supply is cut off; they become intensely congested, swollen and sometimes gangrenous.

Internal haemorrhoids are treated either by injection or excision. Injection gives good results in early cases where bleeding is the main symptom; excision is required for haemorrhoids which prolapse and do not go back spontaneously. Strangulated haemorrhoids are treated initially by fomentations or are replaced under general anaesthesia; later, when the swelling and inflammation have subsided, excision may be undertaken.

## Arteries

### Injuries

Arterial injuries may be classified as follows:

1. Contusion.
2. Rupture, without external wound.
  - (1) Partial.
  - (2) Complete.
3. Division, in association with an external wound.

*Contusion* of an artery may lead to local thrombosis and, in addition, to widespread arterial spasm. These processes, especially the latter, may be sufficient to cause gangrene if they are not relieved.

*Partial rupture*—that is, rupture of the inner and middle coats of an artery—leads to thrombosis; later an aneurysm (page 225) may develop at the site of injury.

*Complete rupture* leads to the formation of a pulsating haematoma; if this is untreated it may either rupture or develop into an aneurysm. General signs of internal haemorrhage may be present, and distal to the injury there may be gangrene or other evidence of impaired arterial blood supply.

*Division of an artery due to a wound* commonly results in severe haemorrhage; sometimes, however, if the artery is completely divided, bleeding

ceases spontaneously as a result of contraction of the media, retraction of the intima and subsequent thrombosis. If the corresponding vein is injured at the same time an abnormal communication may develop between artery and vein. This is known as an arteriovenous aneurysm. Distal signs of impaired arterial circulation may occur as in the case of arterial rupture.

The usual treatment of a ruptured or divided artery is to ligate it above and below the injured portion. Occasionally, if special precautions—including the use of heparin and other anticoagulant drugs—are taken to prevent thrombosis, it is possible to restore the circulation by resecting the damaged segment and reconstituting the vessel by end to end anastomosis or by arterial grafting.

### Chronic obliterative arterial disease

The term chronic obliterative arterial disease includes *atherosclerosis*, arterial narrowing associated with diabetes, and *thromboangiitis obliterans* (often called Buerger's disease).

#### *Atherosclerosis*

In atherosclerosis lipoid material is deposited in the intimal coat of arteries causing narrowing of the lumen, and subsequently thrombosis may occur and completely occlude the vessel. The condition is common in people over the age of 50, but may sometimes be seen in quite young adults. It is often widespread but the extent to which different vessels are affected may vary greatly. Fortunately, in many parts of the body the blood supply is derived from more than one source, and when one artery is occluded others, termed collaterals, may maintain an adequate circulation; if, however, there is no alternative blood supply, or if the collateral vessels are themselves diseased, evidence of *ischaemia* will develop.

The vessels of the lower extremities are commonly affected, and the symptoms and signs may then include intermittent claudication (that is, pain in the muscles brought on by exercise and relieved by rest), coldness and blueness of the skin, absence of arterial pulsation and not infrequently gangrene. Visceral vessels may also be affected, however, including those supplying the brain, heart, kidney and bowel, and the patient's life may then be seriously threatened.

There is at present no cure for atherosclerosis nor any certain way of preventing it, but the patient's symptoms can often be considerably relieved by treatment. The procedures available include: (a) conservative measures such as general care of the affected part, special exercises, and administration of anticoagulants and vasodilators; (b) *sympathectomy*



(Chapter II); (c) arterial surgery including *thromboendarterectomy* and *arterial grafting*; (d) amputation.

Administration of vasodilators and sympathectomy have no effect on grossly atheromatous arteries, but may improve the circulation by causing dilation of relatively healthy collaterals. Sympathectomy may also help patients with threatened gangrene by diverting blood from muscles to skin and subcutaneous tissue. Arterial surgery may be of great value but only if the patients have been carefully selected in the light of special investigations including *arteriography* (Fig. 76—see also Chapter I).

*Thromboangiitis obliterans*

Thromboangiitis obliterans is a rare disease which is confined to males and usually begins in the twenties or early thirties. The patient often



FIG. 76. Arteriogram showing a block in the femoral artery.

experiences attacks of inflammation in superficial veins, and then develops evidence of ischaemia due to pathological changes associated with narrowing of the lumen and subsequent thrombosis in arteries of the extremities. There are often periods of remission and exacerbation, but many patients eventually lose one or both legs. The disease rarely if ever occurs in people who have never smoked and all affected patients should be strongly advised to abstain from smoking.

### Embolism

Embolism is defined as the carriage of a foreign body in the blood stream, and its impaction at a point in an artery where the lumen is too small to permit its further passage. Emboli may be classified as simple, infective and neoplastic.

#### *Simple emboli*

Simple emboli consists of pieces of detached thrombus, fat or air bubbles.

In venous thrombosis, as already described, a piece of clot may become detached and travel via the right auricle and right ventricle of the heart to the pulmonary artery causing *pulmonary* embolism (Chapter II).

In *auricular fibrillation* clot may form in the auricles of the heart. If a piece of clot in the left auricle is detached it travels via the mitral valve, left ventricle and aorta to one of the systemic arteries, and causes systemic embolism. If, as commonly happens, one of the main arteries of a limb is blocked the part of the limb distal to the block is suddenly deprived of its blood supply. The patient complains of sudden severe pain, and the limb becomes pale, pulseless and paralysed. If there is no general arterial disease collateral vessels may open up and restore an adequate circulation; should this not occur gangrene follows.

There are four possible lines of treatment in systemic embolism affecting a limb:

1. *Conservative treatment.* This is the same as the treatment of thrombosis; that is, anticoagulant drugs are given and the limb is kept cool. This procedure is often successful in embolism of the arteries of the upper limb but is unreliable when a large vessel in the lower limb is affected.

2. *Conservative treatment combined with some procedure designed to block temporarily the sympathetic nerve supply to the limb and thus increase the blood flow by dilating collateral vessels.* The usual procedure is to inject a local anaesthetic in the region of the lumbar part of the sympathetic trunk if the lower limb is affected, and round the trunks of the brachial plexus if the upper limb is affected.

3. *Embolectomy*; that is, exposure of the affected vessel and removal of the embolus. To be effective this operation must be performed soon after embolism has occurred. Results are usually good if less than 6 hours have elapsed; after 12 hours, on the other hand, success is unlikely. During the operation the wound is flooded with heparin solution and heparin is administered intravenously for some days afterwards.

4. *Amputation*. If gangrene occurs amputation will be required.

Fat embolism (Chapter VI) and air embolism (page 217) have already been described.

### *Infected emboli*

Infected emboli may be formed by pieces of infected thrombus, or by vegetations from the heart valves in *bacterial endocarditis*. Multiple infected emboli are responsible for *pyaemia* (Chapter X).

### *Neoplastic emboli*

Emboli consisting of living tumour cells are responsible for the metastasis of malignant tumours by the blood stream.

## **Raynaud's phenomenon**

It is common experience that in response to severe cold the fingers and toes become first blue and then waxy white. In some people similar changes occur in response to quite mild degrees of cold or sometimes in response to emotional stimuli, and may be followed in time by gangrene, especially of the tips of the fingers. This condition was first described by a French doctor, Maurice Raynaud, over 100 years ago, and became known as Raynaud's disease. It is, however, not a single disease entity, and it is now customary to distinguish between *Primary Raynaud's disease*, which is a rare condition of unknown etiology occurring particularly in adolescent girls, and *Raynaud's phenomenon* (sometimes called *Secondary Raynaud's disease*) in which similar symptoms develop as a result of a variety of causes including obliterative arterial disease, and a number of others such as scleroderma, cervical rib and the repeated use of vibrating tools which need not greatly concern the dental student.

Patients with primary Raynaud's disease may be greatly improved by the operation of cervico-dorsal-sympathectomy. In Raynaud's phenomenon the underlying condition should be treated if possible; failing this the patient may gain considerable relief from sympathectomy but often relapses after about a year.

### Temporal arteritis

This is a condition of unknown etiology occurring in middle-aged and elderly people, especially women, in which the temporal artery becomes thickened and tender. The overlying skin is often red and the patient may complain of headache and fever.

Administration of corticosteroids may be helpful, but in some patients it is necessary to remove a segment of the affected artery.

### Aneurysm

An aneurysm is a pathological dilatation of an artery. Aneurysms may be congenital or may result from arterial injury or disease.

*Congenital aneurysms* are always small and are important only when they are situated on the arteries of the circle of Willis inside the skull. Spontaneous rupture then sometimes occurs and causes sudden intense headache followed rapidly by loss of consciousness and often by death.

*Aneurysms due to arterial injury* are rare in civilian practice, but fairly common in war surgery as a result of gun-shot wounds.

*Aneurysms due to arterial disease* are nowadays usually atherosclerotic but sometimes still result from syphilis. Atherosclerotic aneurysms occur mainly in the lower part of the abdominal aorta and in the peripheral arteries; syphilitic aneurysms occur characteristically in the thoracic aorta but may also develop in other vessels.

The symptoms and signs depend on the site. In many cases the aneurysm may be felt as a smooth rounded or fusiform swelling which exhibits expansile pulsation (Chapter I). In addition, there may be impairment of the circulation distal to the aneurysm (for example, in the arm with aneurysm of the subclavian artery), and pain or muscular paralysis due to pressure on nerves. Sometimes death occurs suddenly from haemorrhage due to spontaneous rupture of the aneurysm.

The surgical treatment of aneurysms is beyond the scope of this book.

### Haemangiomata

The term *haemangioma*, strictly speaking, means a tumour arising from blood vessels, but most lesions so described are congenital malformation and not true tumours.

Haemangiomata are often classified as *capillary*, *cavernous* (venous) or *arterial*, but many haemangiomata contain both capillary and cavernous elements. Clinically, haemangiomata may be divided into those involving the skin and superficial tissues, and those arising in the liver,

bones and other deep structures. The former are common; the latter are rare and will not be considered.

### **Haemangiomata of the skin and superficial tissues**

Haemangiomata of the skin and superficial tissues may be subdivided into five clinical types.

1. *Telangiectasis*. Unlike the other haemangiomata to be described, telangiectases or 'spider naevi' are not congenital malformations, but result from dilatation of normal capillaries. These dilated capillaries appear as fine radiating red lines. Telangiectases may occur on the face in normal people, especially in middle and old age; they are particularly common in patients with cirrhosis of the liver. They are also common in areas of skin which have been irradiated.

2. *The Port-wine stain*. The port-wine stain is a capillary haemangioma involving the superficial part of the skin. It forms an ugly, flat, reddish-purple lesion which often covers a wide area and is commonly seen on the face (Fig. 77). It is present at birth and grows only in proportion to the growth of the child.



FIG. 77. Port wine stain.

Treatment is difficult. Excision in multiple stages (that is, by a series of operations at each of which only a small part of the lesion is removed) is sometimes possible, but large port-wine stains are usually best left alone. In girls the disfigurement may be minimized by using a special type of make-up known as 'Covermark'.

3. *The strawberry mark or hypertrophic haemangioma.* The strawberry mark is a capillary or mixed capillary-cavernous haemangioma which involves the whole thickness of the skin. It forms a raised bright-red swelling (Fig. 78) which blanches on pressure. The size ranges from a few millimetres to about 3 centimetres in diameter.



FIG. 78. Strawberry mark on the upper lip in an infant.

Strawberry marks may be successfully treated by any of the following methods: application of carbon dioxide snow, injection of a sclerosing fluid, diathermy, radiotherapy and surgical excision. Sometimes these marks disappear spontaneously, however, and except for cosmetic reasons treatment is *not necessary* unless the lesion is increasing in size or threatens to ulcerate.

4. *Subcutaneous haemangioma.* Haemangiomata of cavernous or mixed capillary-cavernous type occur in the subcutaneous tissue, including the subcutaneous tissue of the lip. The lesion may or may not involve the overlying skin.

A purely subcutaneous haemangioma forms a bluish, somewhat spongy, swelling. Treatment is by irradiation or excision. Irradiation is usually best for haemangiomata involving the lip and gives excellent results if small doses of X-rays are given at intervals over a period of several months.

5. *Cirroid aneurysm*. Cirroid aneurysm is rare, but occurs occasionally in the scalp. It consists of dilated tortuous arteries and forms a highly vascular pulsatile swelling. The treatment is excision. The operation is performed in two stages to minimize the risk of severe haemorrhage.

## Chapter XVIII

# DISEASES OF THE LYMPHATIC SYSTEM

### WITH SPECIAL REFERENCE TO THE LYMPH NODES OF THE HEAD AND NECK

Most of the tissues of the body are permeated by a network of fine vessels known as *lymphatics*. From this network larger vessels arise which empty finally via the thoracic and right lymphatic ducts into the great veins in the root of the neck.

The lymphatics play an important part in the interchange of fluid between the blood and the tissues. Fluid reaches the tissues by transudation through the walls of the capillaries; it is removed partly by reabsorption into the capillaries and partly by the lymphatics. Fluid reabsorbed by the capillaries contains very little protein; fluid removed by the lymphatics, known as *lymph*, has approximately the same composition as blood plasma.

Lymphatics are not present in the central nervous system or in avascular tissues such as cartilage, and it is doubtful whether they exist in bone and skeletal muscle.

Along the course of the lymphatics and lymph trunks are situated numerous *lymph nodes*. These nodes are commonly termed lymph glands; they are, however, not glands in the ordinary sense of the word, but form part of the reticulo-endothelial system of the body. Their functions are as follows:

1. To filter the lymph and remove bacteria or other foreign material.
2. To form lymphocytes and probably also macrophages.
3. To form antibodies of various kinds when micro-organisms or foreign proteins have been introduced into the body.

Tissue similar in structure to that found in lymph nodes also occurs in other situations; for example, in the tonsils.

### The Lymphatics and Lymph Nodes of the Head and Neck

The lymph nodes of the head and neck (Fig. 79) may be subdivided into three groups as follows:

1. *The circular chain of nodes*. This consists of the following groups:
  - (1) *Occipital*, between the mastoid process on each side and the external occipital protuberance.
  - (2) *Posterior auricular*, overlying the mastoid process.
  - (3) *Pre-auricular*, a single node lying immediately in front of the tragus.



- (4) *Parotid*, some lying on or just beneath the parotid fascia, and others in the substance of the gland.
- (5) *Facial*, lying along the course of the facial artery.
- (6) *Submental*, situated close to the midline in the submental triangle.
- (7) *Submaxillary*, some situated in close relation to, and others actually in the substance of, the submaxillary salivary gland.

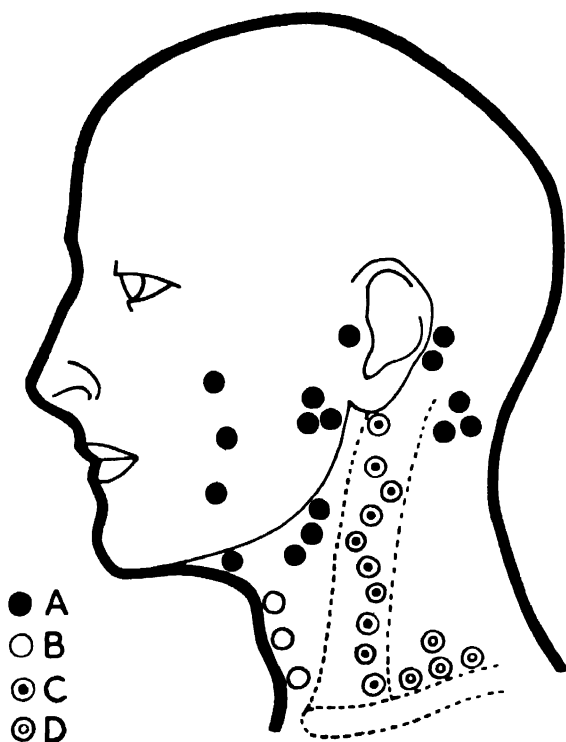


FIG. 79. The lymph nodes of the head and neck.

- A. Circular chain.                      B. Anterior jugular nodes.  
C. Jugular chain.                        D. Supraclavicular nodes.

(8) *Superficial cervical*, lying superficial to the upper part of the sternomastoid muscle, alongside the external jugular vein.

2. *The jugular chain of deep cervical nodes*. The nodes of this chain are situated close to the internal jugular vein and extend from the base of the skull to the root of the neck. Most of them lie under cover of the sternomastoid muscle, but when enlarged they extend forward from beneath its anterior border. Some of the lower nodes, however, lie behind the posterior border of the muscle in the posterior triangle of the neck. These lowermost nodes are sometimes spoken of as 'supraclavicular'.

3. *The anterior cervical nodes.* These are situated close to the midline anterior to the larynx and trachea.

It is unnecessary for our present purpose to describe fully the lymph drainage of the head and neck, but the following facts are of clinical importance and should be remembered.

*The lymph drainage of the scalp*

The posterior part of the scalp drains to the occipital nodes; the side of the scalp to the post-auricular and pre-auricular nodes; and the anterior part to the facial, parotid and pre-auricular nodes. These, in their turn, drain to the upper nodes of the jugular chain.

Infections of the scalp are common and when they occur the corresponding lymph nodes are usually enlarged and tender.

*The lymph drainage of the face and lips*

The face drains primarily to the facial and pre-auricular nodes, and thence to the submental, submaxillary and upper jugular nodes.

In patients with inflammatory lesions on the cheeks the corresponding pre-auricular nodes are usually enlarged and tender.

In patients with epitheliomata of the lower lip metastasis occurs first in the submental and submaxillary nodes, but later the upper members of the jugular chains may become involved.

*The lymph drainage of the tongue and floor of the mouth*

From the apex of the tongue drainage occurs in two directions: to the submental nodes, and to a node of the jugular chain situated fairly low down in the neck.

From the side of the anterior part of the tongue, and the related part of the floor of the mouth, drainage occurs to the submaxillary and upper jugular nodes of the same side.

From the central and posterior parts of the tongue drainage occurs to the jugular nodes of *both* sides of the neck.

The important clinical consequences of these anatomical facts are, first, that in every case of carcinoma of the tongue the submental, submaxillary and jugular nodes of both sides of the neck must be palpated; and secondly, that in every case of enlarged cervical nodes the tongue and floor of the mouth should be carefully examined.

*The lymph drainage of the pharynx*

The lymph drainage of the pharynx need not be considered in detail, but it is important to remember that secondary deposits in the jugular nodes

may be the first indication of a primary pharyngeal carcinoma. Therefore, if fixed, hard nodes are found in the neck, and there is no primary growth on the lip, tongue or the floor of the mouth, a search must be made in the pharynx, including the pyriform fossae. This examination requires special instruments and is best undertaken by an ear, nose and throat surgeon.

### **Diseases of Lymph Nodes**

Enlargement of lymph nodes may be due to many causes, the most important of which are as follows:

1. Acute lymphadenitis.
2. Generalized infectious diseases, including glandular fever, rubella ('German measles') and secondary syphilis.
3. Tuberculous lymphadenitis.
4. Non-specific chronic lymphadenitis.
5. Secondary carcinoma.
6. Hodgkin's disease.
7. Lymphatic leukaemia.
- 8 Lymphosarcoma.

Acute lymphadenitis has been described in Chapter X. It is important to remember that the lymph node enlargement may persist after the primary focus of infection has healed.

Glandular fever and rubella are mainly of medical interest. In both conditions there is fever and widespread lymph node enlargement. Glandular fever is diagnosed by changes in the differential leucocyte count and by a specific serological test; rubella by the characteristic rash.

Secondary syphilis, tuberculous lymphadenitis and non-specific chronic lymphadenitis have been described in Chapter XIII; secondary carcinoma of lymph nodes has been described in Chapter XIV. The problem of differential diagnosis will be considered later.

### **Hodgkin's disease**

Hodgkin's disease is a progressive and fatal disease of unknown etiology in which characteristic changes occur in the lymph nodes and other parts of the reticuloendothelial system.

The disease may occur at any age, but is most common in young adults. It is twice as common in males as females.

The first symptom is usually painless enlargement of a group of lymph nodes, usually in the lower cervical region. The nodes gradually increase in size but remain discrete (Fig. 80). They have a characteristic 'rubbery' consistency.

After a time other nodes are affected (for example, in the axillae and groins) and enlargement of the spleen and liver may occur. Constitutional symptoms such as fever and lassitude may develop, and the blood count shows anaemia and often eosinophilia. The histological changes in the nodes and elsewhere are usually characteristic; they include proliferation of reticulum and endothelial cells with the formation of giant cells, and accumulation of eosinophils and plasma cells.

*Treatment.* The disease cannot be cured, but X-ray therapy may prolong life for several years, especially in patients who contract the disease in middle or old age.



FIG. 80. Enlarged cervical lymph nodes due to Hodgkin's disease.

### **Lymphatic leukaemia**

Lymphatic leukaemia is a fatal disease in which there is widespread hyperplasia of lymphoid tissue associated with changes in the circulating blood; these include a great increase in the number of lymphocytes per cu.mm., the presence of primitive cells of the lymphoblast type and some degree of secondary anaemia.

There are two forms of the disease: acute and chronic.

Acute leukaemia may occur at any age, but is most common in children. Lymph nodes and lymphoid tissue in various parts of the body become enlarged, haemorrhages occur subcutaneously and from mucous membranes, and death supervenes within a few days or weeks. Chronic lymphatic leukaemia occurs only in adults, usually between the ages of 50 and 60. There is widespread lymph node enlargement and usually also some

enlargement of the spleen. The affected nodes are discrete, and moderately firm in consistency; they are rarely as big as those found in Hodgkin's disease and lymphosarcoma (*vide infra*). The patient may survive for 2 or 3 years, but dies eventually from cachexia, haemorrhage or intercurrent infection. The diagnosis is made by examination of the blood, aided if necessary by biopsy of a node.

Treatment at present is only palliative. For this purpose X-ray therapy is most widely used, but in recent times encouraging results have been obtained with certain drugs, including nitrogen-mustard (a derivative of mustard gas) and urethane.

### **Lymphosarcoma**

Lymphosarcoma is a rare malignant tumour which occurs in lymph nodes and in masses of lymphoid tissue such as the tonsil. It develops usually between the ages of 30 and 60, and is commoner in men than in women.

The tumour does not remain localized, but soon breaks through the capsule to involve the surrounding tissue; it also spreads to other nodes in the vicinity. In this way a large, hard, irregular swelling is formed. After a short time similar tumours develop in nodes elsewhere in the body, the patient becomes anaemic and cachectic, and death occurs usually within a few months. The diagnosis is suggested by the history and clinical findings, and confirmed by biopsy. Histologically the tumour consists of masses of immature lymphocytes.

*Treatment.* X-ray therapy causes dramatic diminution in the size of the tumours or even their complete disappearance, but this remission, unfortunately, is only temporary.

### **The differential diagnosis of enlarged cervical lymph nodes**

Enlarged lymph nodes occur so commonly that with every swelling in the neck the first question to ask oneself is: 'Does it arise in lymph nodes?' If so, the next problem is to determine the pathological nature of the swelling.

The origin of a cervical swelling may be apparent from its position, but in many cases a full investigation is necessary before a complete diagnosis can be made.

Begin by taking a careful history and making a thorough clinical examination (Chapter II). The following features are of special importance:

1. The age of the patient.
2. The length of history.
3. The position and character of the swelling.
4. The presence (or absence) of a lesion in the area drained by the nodes.

5. The presence (or absence) of enlarged nodes in other parts of the body and of enlargement of the spleen and liver.

6. The presence (or absence) of fever and other constitutional symptoms and signs.

If the diagnosis remains in doubt special investigations must be undertaken. These include:

1. Differential leucocyte count.

2. Bacteriological and serological tests (for example, the Mantoux and Wassermann tests).

3. Biopsy.

Biopsy of a lymph node is usually a simple procedure and should be undertaken whenever a diagnosis cannot be reached by other means.

The important diagnostic features of lymph node swellings in the neck may be summarized as follows:

In *acute lymphadenitis* the swelling has usually been present for only a few days. It is painful and tender, and the overlying skin is often reddened; if an abscess has formed fluctuation may be demonstrated. An acute inflammatory lesion is usually present in the area drained by the nodes. The temperature is moderately raised (for example, 101° F.).

Lymph node enlargement due to *glandular fever* and *secondary syphilis* is not confined to the neck. The nodes remain discrete and in glandular fever are often tender. The diagnosis is confirmed by finding other evidence of the disease (for example, the characteristic rash in secondary syphilis) and by special tests (the differential leucocyte count and a specific serological test in glandular fever; the Wassermann reaction in syphilis).

*Tuberculous cervical lymphadenitis*, as we have seen (Chapter XIII), is now rare, but before pasteurization of milk and tuberculin testing of cattle were widely practised it was quite common, especially in children and young adults. Except in early cases the nodes are matted together, and may show evidence of softening and involvement of the skin. The upper nodes of the jugular chain and the submaxillary nodes are the ones most commonly affected.

Breaking down tuberculous nodes in the upper part of the jugular chain may be mistaken for a *branchial cyst*. If a collar stud abscess has formed there will be involvement of the skin and no difficulty should arise; if, however, the cold abscess is still deep to the deep fascia the diagnosis may remain in doubt until some of the contents of the swelling are aspirated and examined (Chapter I).

Tuberculous submaxillary nodes must be distinguished from a *swelling of the submaxillary salivary gland*. Difficulty arises because the affected nodes may lie within the capsule of the gland and when enlarged may give

rise to a swelling which is palpable bimanually (Fig. 7). A swelling of the gland due to a calculus in Wharton's duct (Chapter XXIII) usually increases in size when the patient takes food. The calculus may be visible at the orifice of Wharton's duct; if situated further back it may be palpated or demonstrated radiologically. Calculi within the gland cause chronic suppuration. Pus may be seen to enter the mouth when the gland is gently compressed, and a radiograph reveals the calculi. Swelling of the sub-maxillary salivary gland due to other causes is rare and biopsy is usually necessary to make the diagnosis.

Occasionally tuberculosis occurs in the pre-auricular or parotid nodes; care is then required to distinguish the swelling from a *mixed parotid tumour* (Chapter XIV). In mixed parotid tumour, however, there is no softening or involvement of the skin despite the fact that the swelling has usually developed slowly over a period of months or years, and nodes elsewhere in the neck are not enlarged.

*Chronic non-specific adenitis* resembles early tuberculous adenitis but the nodes do not show the same tendency to break down and the inflammation usually resolves in a few weeks. There may be a primary focus of infection but this has often healed before the patient reports with a swelling in the neck.

In *Hodgkin's disease* the nodes have a rubbery consistency and even when very large remain discrete. At first the disease may be confined to the neck; later nodes elsewhere are affected and the spleen may also be enlarged. Biopsy is usually performed to confirm the diagnosis.

In *lymphatic leukaemia* there is widespread lymph node enlargement. The nodes are discrete and not tender. The diagnosis rests on examination of the blood, aided, if necessary, by biopsy.

In *lymphosarcoma* there is a rapidly growing swelling which infiltrates other structures in the neck. Nodes elsewhere soon become enlarged. The diagnosis depends on biopsy.

*Secondary carcinoma* is the commonest cause of a chronic lymph node swelling in the neck in a patient over the age of 50. The swelling is very hard and, except in early cases, fixed. The primary lesion can usually be found if looked for, but occasionally it is hidden in a relatively inaccessible region. If the primary growth is not found a biopsy should be performed on the swelling in the neck.

### **Diseases of the Lymphatic Vessels**

*Inflammation* of the walls of lymphatic vessels is called *lymphangitis*. Acute lymphangitis, which is the common type, has been described in Chapter X.

Obstruction of lymphatic vessels may be produced in several ways; for example, by tumour cells, or as the result of infestation with a parasite (*Filaria sanguinis hominis*) which is common in certain tropical regions.

Lymphatic obstruction often causes oedema. Gross obstruction, such as occurs in many cases of filarial infestation, results in the condition known as *elephantiasis*.

### Lymphangioma

Strictly speaking the term lymphangioma means a tumour of lymphatic vessels, but it is commonly used to denote any localized swelling resulting from overgrowth or dilatation of lymphatics. Most of these swellings are not true tumours but congenital malformations. In the head and neck they usually occur in one of the following sites:

1. In the tongue, forming one variety of *macroglossia*.
2. In the lip, forming one variety of *macrocheilia*.
3. In the neck, forming a soft, fluctuant and translucent swelling known as a *cystic hygroma* (Fig. 81).



FIG. 81. Cystic hygroma.

Lymphangiomata frequently do not require treatment. If they are unsightly or if, as often happens, they are the site of recurring attacks of inflammation, they should be excised. Owing to the diffuse nature of the swelling, complete excision may be difficult or impossible.



## *Chapter XIX*

# DISEASES OF BONE

The diseases of bone which are of importance to dental students may be classified as follows:

1. *Inflammatory conditions*
  - (1) Osteomyelitis.
  - (2) Tuberculosis.
  - (3) Syphilis.
2. *Tumours of bone*
  - (1) Benign tumours.
  - (2) Malignant tumours.
    - (i) Primary.
    - (ii) Secondary.
3. *Bone cysts*
4. *Miscellaneous conditions*
  - (1) Congenital disorders.
  - (2) Disuse atrophy.
  - (3) Deficiency diseases affecting bone.
    - (i) Rickets.
    - (ii) Scurvy.
  - (4) Endocrine disorders affecting bone.
    - (i) Generalized osteitis fibrosa cystica.
    - (ii) Gigantism and acromegaly.
  - (5) Paget's disease.
  - (6) Senile osteoporosis.

### **Osteomyelitis**

#### **Acute osteomyelitis**

Acute osteomyelitis (that is, acute suppurative inflammation of bone) arises in one of three ways:

1. *By blood-borne infection from a focus elsewhere in the body.* Organisms enter the blood stream, often as the result of a trivial abrasion, sometimes from a focus of suppuration such as a boil or carbuncle. Bacteriaemia or septicaemia (Chapter X) results, and from the blood organisms reach the bone.

This form of osteomyelitis occurs in the long bones of children and adolescents prior to the time of fusion of the epiphyses; it is more common in boys than girls. The causal organism is usually a *staphylococcus*.

The disease usually begins in the *metaphysis*; that is, the part of the shaft immediately adjacent to the epiphysis. Pus forms and spreads inwards to the medullary cavity and outwards to form a collection beneath the periosteum; it may then extend longitudinally, stripping the periosteum from the bone. Spread to the nearby joint is rare except when the epiphysis is intracapsular as in the hip joint; when it occurs suppurative arthritis results.

In adults acute blood-borne osteomyelitis occurs in the spine and pelvis, but the long bones are rarely affected.

2. *As the result of a compound fracture.* Here the osteomyelitis is merely part, though often the most serious part, of a wound infection. The causal organism is usually a staphylococcus, sometimes a streptococcus and occasionally one of the other pyogenic bacteria. A mixed infection occurs in many cases.

3. *By direct spread of pyogenic infection to the bone from a nearby focus.* Osteomyelitis of a rib, for example, may occur as a result of drainage of an empyema by rib resection; and osteomyelitis of the mandible (*vide infra*) as a complication of dental infection.

#### *Special features of acute inflammation in bone*

The blood vessels which supply bone run in rigid tubes—the Haversian canals—and as a result, when inflammatory exudation occurs, the vessels are subjected to pressure and the blood stream is slowed and finally brought to a standstill by thrombosis. Portions of bone may thus be deprived of their blood supply and undergo necrosis. A piece of dead bone is termed a *sequestrum*. It cannot be absorbed, and must be removed or discharged to the surface spontaneously before healing will occur. A certain amount of new bone may be laid down around a sequestrum forming an *involucrum* or 'new case'. Holes known as *cloacae*, which give egress to pus, are commonly seen in an involucrum.

In many cases the organism is sensitive to penicillin and *early* treatment with this drug often leads to resolution without the formation of a sequestrum. In subacute pyogenic infections of bone there may be gradual erosion without death *en masse*; this condition is called *caries*. Caries occurs more commonly in tuberculosis of bone (Chapter XIII), however, than in osteomyelitis.

*Symptoms and signs of acute osteomyelitis*

In the early stages the only local symptom may be rather ill-defined and fleeting pain in a limb; soon, however, the affected bone becomes exquisitely tender and the overlying tissues may show evidence of acute inflammation. In the later stages one or more sinuses may be present; these discharge pus and usually lead down to a sequestrum. Exuberant granulation tissue, known to the laity as 'proud flesh', is often seen at the orifice of the sinus. The neighbouring joint is sometimes distended—usually as a result of a so-called 'sympathetic' serous effusion, occasionally because of suppurative arthritis.

The constitutional symptoms and signs are often severe, especially in blood-borne osteomyelitis, as this condition can occur only as a result of bacteraemia or septicaemia.

X-ray usually reveals no abnormality for 2 or 3 weeks; thereafter it may show decalcification of the affected bone. Later there may be evidence of new bone formation and a sequestrum may be seen; the latter appears as a piece of dense bone surrounded by an area of rarefaction (Fig. 82).

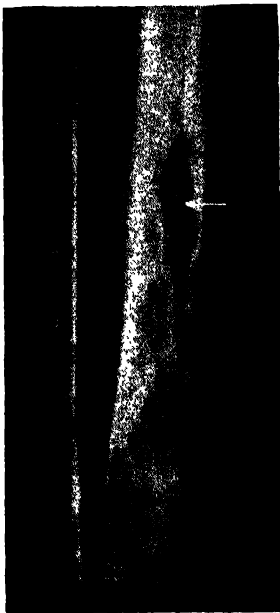


FIG. 82. Radiograph in a case of osteomyelitis showing much new bone formation and a sequestrum (marked by arrow).

*Treatment*

The advent of antibiotics has revolutionized the prognosis and treatment of acute osteomyelitis.

The patient is put to bed and a sample of venous blood is taken under sterile conditions for bacterial culture. This is found to be positive in about 50 per cent of cases. The affected limb is splinted and the usual general treatment for serious acute inflammation is instituted, including injections of penicillin in full dosage. If the infection is not controlled by penicillin, or if the blood culture yields organisms which are not penicillin sensitive, another antibiotic is substituted. In the absence of a positive blood culture erythromycin or methicillin may be tried.

Incision may be required for evacuation of a subperiosteal collection of pus, and occasionally holes are drilled into the bone to reduce tension in the medullary cavity. Extensive operations on the bone are not indicated during the acute stage of the disease.

If a sequestrum forms it will have to be removed. The operation is postponed until the sequestrum has become completely free; this is recognized by radiological examination and by probing through a sinus.

### **Chronic osteomyelitis**

Chronic pyogenic osteomyelitis occurs in two forms:

1. *Persistence of chronic inflammation in a bone which has been the site of acute osteomyelitis.*

This condition is quite common. Constitutional symptoms are mild or absent, but over the affected bone there are one or more sinuses which discharge pus and sometimes small sequestra. Sometimes the patient has recurring attacks of acute inflammation ('flares') during which the bone becomes tender and the overlying skin becomes red and oedematous.

The only satisfactory treatment is to 'gutter' the bone and remove, as far as possible, all diseased tissue. Formerly the wound was left widely open until it was clean and granulating, when a split skin graft was applied; nowadays, however, if an appropriate antibiotic is given systemically, it is often possible to close the wound by primary suture.

2. *Brodie's abscess.*

This is a chronic abscess which occurs near the end of a long bone. The infection is blood borne. Sometimes a Brodie's abscess develops years after an attack of acute osteomyelitis; it is then thought to be due to organisms carried to the site during the septicaemic phase of the acute disease. In other cases the infection has come from some trivial and unremembered lesion.

The organisms remain dormant for a long time and then become active, sometimes as a result of injury. When this happens the patient complains of pain and the overlying skin may become inflamed. There is usually a visible swelling in the bone, and X-ray shows a translucent cavity which is surrounded by dense (sclerosed) bone and sometimes contains a sequestrum.

The treatment is to open the abscess by incising the soft tissues and chiselling away the bony wall until only a shallow saucerized hollow remains. The wound is then lightly packed. Penicillin is given before and after operation.

### **Osteomyelitis of the mandible**

Osteomyelitis of the mandible is usually due to the spread of apical or periapical infection; less commonly it develops as a complication of a fracture. Other causes, including blood-borne infection, are rare. We shall consider only osteomyelitis of dental origin.

In many patients the spread of infection from a tooth gives rise to an alveolar abscess (Chapter X); in a few patients osteomyelitis develops. *The danger of bone infection occurring is increased by unskilful dental extraction and by the use of local analgesia in the presence of infection.*

Clinically osteomyelitis of the mandible is of two kinds: diffuse and localized.

*Diffuse osteomyelitis* of the mandible may occur at any age, but is commonest between 20 and 40. Males and females are affected equally. In most cases there is a history of recent dental extraction. The condition is often described as acute but, as Wass has pointed out, this is misleading. In contradistinction to acute osteomyelitis of a long bone, diffuse osteomyelitis of the mandible is not associated with septicaemia or pyaemia and the constitutional symptoms are mild.

The patient complains of constant boring pain, and there is swelling due to periosteal thickening or to cellulitis of the overlying soft tissues. Trismus may be present if the molar region of the jaw is involved. In many cases there is a discharge of pus into the mouth through a tooth socket or a secondary sinus. An X-ray at first reveals nothing abnormal; after 3 or 4 weeks there is loss of bony detail, and later still sequestra may be seen. A pathological fracture sometimes occurs.

*Localized osteomyelitis* is always chronic. The onset is insidious and the condition may have been present for months before the patient seeks advice. It occurs later than diffuse osteomyelitis, usually after the age of

50, and a history of dental extraction shortly before the onset of symptoms is obtained in only about 50 per cent of cases. Males and females are equally affected.

Pain is less severe than in diffuse osteomyelitis and the swelling is more localized. A radiograph shows one or more areas of rarefaction, sometimes related to a buried root; the surrounding bone is sclerosed and sequestration may be evident. Pathological fracture occurs occasionally.

### *Treatment*

In diffuse osteomyelitis the best results are obtained by combining antibiotic therapy with radical surgery. Initially penicillin should be given in full dosage, but if this fails to control the infection another antibiotic should be substituted. If treatment could be given within 48 hours of onset the disease might be cured without operation, but in practice the diagnosis cannot be made with certainty until radiographic signs develop. By this time operation must be undertaken to remove all diseased bone and relieve tension; failing this, extension of the disease and pathological fracture will occur. An external approach to the bone is usually necessary.

In localized osteomyelitis operation is always required. If only the alveolar margin of the bone is affected the operation may be performed from inside the mouth; in other cases an external approach is used.

### **Osteomyelitis of the maxilla**

Osteomyelitis is much less common in the maxilla than in the mandible, and when it occurs is almost always localized. The reason for this difference is anatomical. The mandible derives almost the whole of its blood supply from a single artery, the inferior dental artery. If this vessel becomes thrombosed the whole bone becomes less resistant to infection, and much of it may sequestrate. The maxilla, on the other hand, receives a series of descending branches from the third part of the maxillary artery, and from the infraorbital artery; the collateral circulation is free and thrombosis in a single vessel is of little consequence.

### **Tuberculosis and Syphilis**

Tuberculous and syphilitic lesions of bone have been described in Chapter XIII.

### **Benign Tumours of Bone**

The benign tumours of bone include chondroma, osteoma and giant-celled tumour (sometimes called osteoclastoma).

**Chondroma**

A chondroma is a benign tumour composed of cartilage. Chondromata may develop from laryngeal or costal cartilage, but more commonly arise from bones. They may project outwards (ecchondromata) or grow in the substance of the bone (enchondromata).

An *ecchondroma* usually arises from a long bone in the region of the metaphysis or from the pelvis. It forms a firm, lobulated, immobile swelling. Ecchondromata often develop before puberty and may stop growing in early adult life. They frequently become ossified and are then called *cancellous osteomata*. Large ecchondromata occasionally undergo malignant change and become *chondrosarcomata*.

*Enchondromata* occur most commonly in the metacarpals and bones of the fingers. They are often multiple. Sometimes the bone is expanded, but often there is no visible swelling and the condition may pass unrecognized unless a pathological fracture occurs. X-ray reveals a clear oval area having a sharp outline. Enchondromata do not undergo malignant change, but in view of the danger of pathological fracture they should usually be removed. If a large cavity remains it may be filled with bone chips.

**Osteoma**

Osteomata are of two types. *Cancellous* osteomata (*vide supra*) occur near the ends of long bones, and *hard* or 'ivory' osteomata arise from the bones of the skull. An ivory osteoma usually forms a hard painless swelling on the lateral aspect of the skull, but sometimes grows inwards into the cranial cavity or the frontal sinus. It is usually best left alone; in the rare cases where there is pressure on the brain the tumour should be removed with a surrounding area of healthy bone, and the defect in the skull repaired with a bone graft or some plastic material.

**Giant-cell tumour**

Giant-cell tumours are highly cellular and contain numerous multinucleated giant cells. They occur in the mouth, where they arise from the peripheral bone of the jaw and form one variety of epulis (Chapter XIV), and near the ends of long bones; similar tumours may also develop from tendon sheaths.

The symptoms of giant-cell tumour of a long bone usually appear in early adult life; they include pain, swelling and sometimes spontaneous fracture. X-ray reveals a rarified area having a lobulated outline.

The treatment is X-ray therapy or surgical excision. X-ray therapy gives good results with giant-cell tumours of long bones; excision is the treatment of choice with myeloid epulis. The tumour must be removed completely otherwise it will recur.

### Malignant Tumours of Bone

#### Primary tumours

Malignant tumours may arise from periosteum, bone or bone marrow. The common malignant tumour of bone is termed 'osteogenic sarcoma'. This name does not imply that these tumours necessarily form new bone, but means that they arise from cells whose normal function is to form bone (osteoblasts). Many osteogenic sarcomata do in fact form new bone but some are purely destructive.

Osteogenic sarcoma is highly malignant and usually metastasizes to the lungs at an early stage. It may arise in any bone, including the jaws, but is commonest in the long bones of the limbs. Children and adolescents are most often affected but the tumour may occur at any age. In older people it sometimes develops in bones which are affected by Paget's disease (*vide infra*).

The first symptom is aching pain in the affected bone, often most severe at night. This is followed by the appearance of a swelling. The diagnosis can usually be made on clinical and radiological grounds (Figs. 83 and 84), but a Wassermann test should always be done because a gummatous lesion in bone sometimes closely resembles a sarcoma.



FIG. 83. Osteogenic sarcoma of the left humerus.



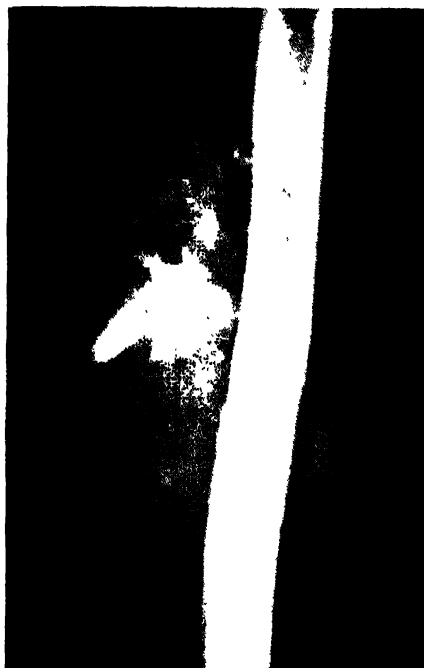


FIG. 84. Radiograph of the tumour shown in Fig. 83.

A radiograph of the chest should always be taken, and if this reveals metastases in the lungs no treatment should be undertaken apart from symptomatic measures for the relief of pain. If the lungs appear clear histological confirmation of the diagnosis must be obtained by biopsy before any form of radical treatment is undertaken.

The prognosis is extremely poor. The best treatment available at the present time is to give a full course of supervoltage X-ray therapy to the tumour and, if the chest remains clear, carry out an amputation a few months later.

*Fibrosarcomata* arising from the periosteum are usually much less malignant than osteogenic sarcomata; they are less invasive and metastasize much later. In consequence, early operation may offer good prospects of cure.

The remaining primary malignant tumours of bone are rare and need not be considered.

### **Secondary tumours**

Metastatic deposits in bone are usually carcinomatous. They are common, and occur most often in the spine, pelvis and long bones of the limbs. The primary growth may be situated anywhere, but carcinomata of the breast,

thyroid, kidney, prostate and lung show a special tendency to metastasize to bone.

Except in superficial bones, such as those of the skull, secondary tumours rarely give rise to palpable swelling. They usually cause dull aching pain, but are sometimes not diagnosed until a routine radiological search is undertaken (Fig. 85) or a pathological fracture occurs (Fig. 86).

Once a malignant tumour has metastasized by the blood stream, whether to bone or elsewhere, no treatment holds out any prospect of cure.



FIG. 85. Radiograph showing secondary carcinomatous deposits in the pelvis. The primary tumour was in the prostate.

### **Malignant growths of the upper jaw**

The term malignant upper jaw includes the following conditions:

1. Invasion of the maxilla by a carcinoma originating in the mucous membrane lining the maxillary antrum or the ethmoidal air cells.
2. Osteogenic sarcoma arising in the maxilla itself.
3. Invasion of the maxilla by a squamous carcinoma originating in the mucous membrane of the mouth or the skin of the face.
4. Invasion of the maxilla by a rodent ulcer of the face.

With carcinoma of the antrum the initial symptoms are usually pain, purulent or blood-stained nasal discharge which is often unilateral, and loosening of the teeth. Later there may be bulging of the cheek (Fig. 87) or hard palate, and epiphora due to blockage of the nasolachrymal duct.



FIG. 86. Radiograph showing a pathological fracture of the humerus due to secondary carcinoma.



FIG. 87



FIG. 88

FIG. 87. Sarcoma of the left maxilla.

FIG. 88. Radiograph of the patient shown in Fig. 87. There is extensive destruction of the alveolus and opacity of the antrum on the left side.

Radiological examination may show opacity of the antrum or destruction of its bony walls (Fig. 88).

The usual treatment is to give X-ray therapy, and later open the antrum by a Caldwell-Luc approach and insert radium on a mould to deal with residual growth. Occasionally early cases are treated by excision of the maxilla.

Sarcoma of the maxilla causes pain and swelling without other symptoms. The treatment is X-ray therapy.

Invasion of the maxilla by carcinomata of the mouth is uncommon. Invasion by a rodent ulcer of the face is also rare nowadays; the condition has been discussed in Chapter XIV.

### **Malignant growths of the lower jaw**

The lower jaw is commonly invaded by a buccal carcinoma. The tumour usually arises from the alveolar mucosa; less commonly it begins in the floor of the mouth, on the tongue or on the lip. The clinical features and treatment of this condition have been described in Chapter XIV.

Other malignant growths of the mandible include secondary carcinomatous deposits, osteogenic sarcoma and malignant adamantinoma. These are all rare, especially the last, and need not be further considered.

### **Bone Cysts**

Bone cysts may be classified as follows:

#### *1. Cysts of dental origin occurring exclusively in the jaws*

These have been described in Chapter XV.

#### *2. Generalized osteitis fibrosa cystica*

In this condition there is generalized decalcification of the bones with multiple cyst formation, due to an adenoma of one of the parathyroid glands (Chapter XXII). The adenoma is rarely palpable, but its presence may be inferred from the changes in the bones, together with an increased calcium content and other changes in the blood.

The treatment is removal of the parathyroid adenoma.

#### *3. Solitary cysts of unknown etiology and localized osteitis fibrosa cystica*

Cysts of unknown etiology occur in the shafts of long bones, most commonly in adolescents and young adults. Sometimes there is a single cavity lined by fibrous tissue and surrounded by normal bone (*solitary cyst*);

less commonly an area of bone is replaced by a mass of fibrous tissue and partly calcified bone containing numerous cystic spaces (*localized osteitis fibrosa cystica*). The blood calcium is normal and there is no abnormality of the parathyroid glands.

The bone may be slightly expanded, but often the first symptom is a pathological fracture. In a radiograph a solitary cyst has a sharp oval outline and often appears trabeculated (Fig. 89); it may be indistinguishable from a giant-cell tumour until a biopsy is performed.



FIG. 89. Radiograph showing a solitary bone cyst in the femur.

A small solitary cyst does not require treatment, but one which is large enough to weaken the bone seriously is best treated surgically before fracture occurs. The lining is curetted out and the cavity packed with chips taken from another bone. If a fracture has occurred it is treated by reduction and immobilization; union nearly always occurs and often the cyst disappears.

Localized osteitis fibrosa cystica is treated by excising the whole diseased area and replacing it by a bone graft.

#### 4. *Hydatid cysts*

Hydatid cysts are very rare in the British Isles and will not be discussed.

### **Congenital Disorders of Bone**

Congenital disorders of bone are not of great importance, but two may be mentioned briefly:

### 1. *Osteogenesis imperfecta*

In osteogenesis imperfecta the bones are weak and brittle, and multiple fractures occur from trivial causes. The victims of this condition usually have blue sclerotics.

The only treatment is to advise the patient to avoid strenuous games and to treat the fractures as they occur. Fortunately, as the patient grows older, the tendency to fracture usually diminishes.

### 2. *Achondroplasia*

Achondroplasia is one of many causes of dwarfism. There is impaired growth of the bones which develop in cartilage; as a result the bones of the limbs are unusually short while the vault of the skull, which develops in membrane, is of normal size.

## **Disuse Atrophy**

Disuse of any part of the body leads to atrophy or, in young subjects, to impairment of growth. The bones are no exception to this general rule. In a paralysed limb, for instance, the bones undergo marked decalcification, and if the paralysis occurs before the epiphyses have fused the limb becomes stunted as compared with the healthy limb on the opposite side. Decalcification may also be caused by immobilization in plaster.

In the jaws atrophy of the alveolar processes occurs after extraction of all the teeth (Fig. 90); it may be minimized by prompt provision of dentures.

## **Rickets**

Rickets is caused by deficiency of Vitamin D. The substance is normally synthesized by the body as a result of the action of ultra-violet light on the skin, and deficiency is usually due to lack of exposure to sunlight.

The disease occurs in infancy and early childhood, but many of its effects persist in later life. Irregular growth occurs at the epiphyses and at the junctions of the ribs and costal cartilages; and the bones become decalcified, soft and pliable so that deformities occur. Later, recalcification takes place, but the deformities remain. The legs show either bowing or knock-knee, the skull becomes broader than usual but flattened in an antero-posterior direction, and distortion of the thorax results in the deformity known as 'pigeon chest'. In addition, gastro-intestinal disturbances occur and the child becomes pale, flabby and pot-bellied.

Rickets may be prevented by regular exposure to sunlight or ultra-violet light from a mercury lamp, or by giving cod liver oil, which is a potent source of Vitamin D.



Fig. 90. No. ku B. Skull of a patient with a subcutaneous tumor.



B. No. 10. Skull of a patient with a subcutaneous tumor.

In older children, when the bones have recalcified, operation is sometimes necessary for the correction of deformities in the limbs.

### **Scurvy**

Scurvy is due to deficiency of Vitamin C, a water-soluble vitamin present in large quantities in most fruits (especially oranges and lemons), in green vegetables and tomatoes, and to a lesser extent in fresh milk. As a result of the deficiency the formation of intercellular substance by cells of the fibroblast type is impaired.

Years ago scurvy occurred frequently in adults, especially in sailors undertaking long voyages; it is now uncommon except in infants.

Clinically the most important manifestations are bleeding spongy gums, and subperiosteal haemorrhages due to rupture of the abnormally weak walls of small blood vessels. The contrast between the purplish colour of the gums and the pallor of the mucous membrane elsewhere in the buccal cavity is striking. It is important to remember that Vincent's infection may supervene in patients with scurvy, and the presence of Vincent's organisms in a smear should not lead one to overlook the possibility of underlying deficiency disease.

Administration of orange juice, tomato juice or pure Vitamin C is effective for either prevention or treatment.

### **Gigantism and Acromegaly**

An adenoma arising from the eosinophil cells of the pituitary gland (Chapter XXII) may cause marked changes in the bones and other tissues.

If the adenoma occurs before the epiphyses of the long bones have fused the bones grow to an abnormal length and gigantism results.

If the adenoma does not develop until after the epiphyses have fused the bones are of normal length, but other changes occur. The bones of the face become thickened, and there is coarsening of the soft tissues particularly of the face and hands. This condition is termed acromegaly; it is recognized by the large horselike face and clumsy spade-like hands (Figs. 91 and 92). The deformity is unpleasant, but the disease has a more serious significance. The adenoma, like other pituitary tumours, is liable to exert pressure on the optic chiasma, causing diminution of the visual fields and ultimately complete blindness. Surgical removal of the tumour is sometimes necessary for this reason, but it is unlikely to lead to improvement in the appearance of the patient.





FIG. 91. Acromegaly.

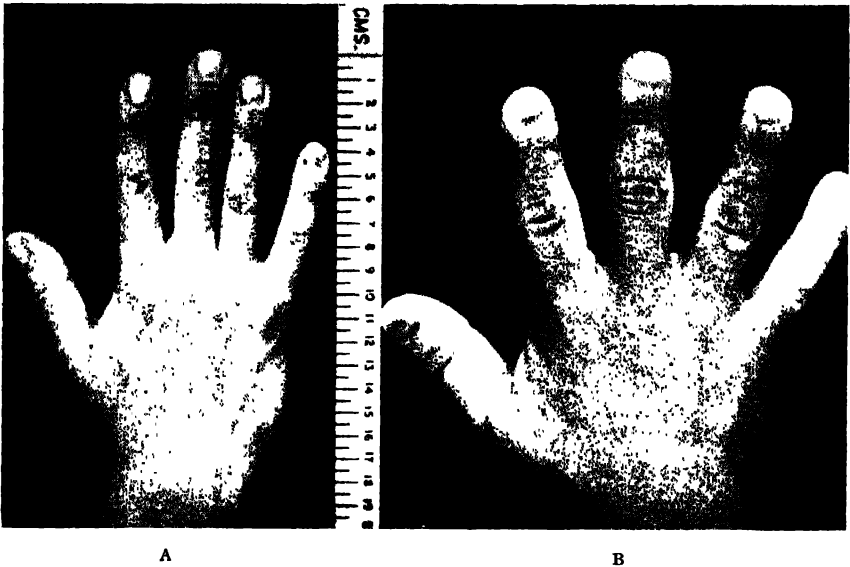


FIG. 92. A. A normal hand.  
B. The large spade-like hand of a patient with acromegaly.

### **Paget's Disease**

Paget's disease of bone occurs in middle and old age, and is much commoner in men than women. The etiology is unknown.

The bones become decalcified, softened and deformed; later they become thickened, and finally recalcification occurs.

Clinically the main features are *kyphosis* (antero-posterior bending of the spine), progressive enlargement of the skull due to thickening of the bone, bowing of the tibiae and femora, and pains in the limbs. The radiological changes are characteristic and are usually seen in the limbs, skull and pelvis.

Pathological fractures are fairly common because the bones remain brittle even after recalcification; they usually unite readily. Sometimes an osteogenic sarcoma develops in one of the affected bones.

Patients with Paget's disease often die of heart failure. It has recently been shown that there are abnormal communications between arteries and veins in the affected bones; because of these an undue strain is imposed on the heart.

No treatment has yet been found which will arrest the progress of the disease, but if pain is severe X-ray therapy often gives symptomatic relief.

## *Chapter XX*

# DISEASES OF JOINTS

### Clinical Examination of Joints

Begin with *inspection*. Note the position in which the joint is held, and whether there is any swelling of the joint or related structures any wasting of the muscles which move the joint, or any bony deformity. Compare the joint to be examined with its fellow on the opposite side but remember that both joints may be the site of injury or disease.

Secondly *palpate* the joint without moving it. Note the position of the bony prominences in the vicinity and if there is any swelling test for fluctuation. Once again compare the affected joint with the corresponding one on the opposite side.

Thirdly, check the previous observations by making appropriate *measurements*. Circumferential measurements provide a quantitative estimate of the degree of swelling or muscular wasting; measurements in the long axis of a limb will reveal shortening due to dislocation or gross disorganization of the joint, but care is necessary to distinguish this from shortening due to a fracture.

Fourthly, test *movements* at the joint. First observe *active movements*—that is, movements carried out by the patient himself; then, if these are limited in any direction, test cautiously the range of *passive* movement. Record any departure from normal, whether it be limitation of movement or abnormal mobility, making use once more of the principle of comparison.

Next, make a *general clinical examination* to determine whether there are lesions in other parts of the body which throw light on the joint condition; for instance, septic teeth in rheumatoid arthritis or signs of *tabes dorsalis* in a suspected neuropathic joint.

Finally, undertake such *special investigations* as seem necessary. These will include *radiological examination* in every case; aspiration of fluid for examination if haemarthrosis or infective arthritis is suspected; estimation of the *blood sedimentation rate* in many joint diseases including tuberculosis; *blood culture* in suppurative arthritis; and the Wassermann (or Kahn) test in cases of suspected syphilis. Occasionally in chronic diseases, if the diagnosis remains in doubt, biopsy of the synovial membrane is indicated.

### Some General Principles of Joint Pathology

The agents which are known to be capable of causing disorders of joints are as follows:

1. Trauma.
  - (1) Acute trauma.
  - (2) Long continued, or recurrent, mild trauma.
2. Infection with micro-organisms.
3. Absorption of bacterial toxins.
4. Metabolic disorders.
5. Certain disorders of the nervous system.

It seems likely, however, that there are other factors, which are at present unknown.

Injury or disease of the synovial membrane often results in an outpouring of clear fluid into the joint. This is known as a *synovial effusion*.

Destruction of articular cartilage causes permanent impairment of function because cartilage has no power of regeneration. If the articular cartilage is destroyed over a wide area *ankylosis* (that is, fixity of the joint) is likely to occur. There are two types of ankylosis: fibrous ankylosis and bony ankylosis. In *fibrous ankylosis* the bones taking part in the articulation are joined together by strong fibrous tissue. Slight movement is often possible and is usually painful. The condition commonly occurs as a result of tuberculous arthritis. In *bony ankylosis* the joint space becomes obliterated by the bones fusing together. No movement is possible and the affected joint is painless. Bony ankylosis commonly results from suppurative arthritis; it may also be produced deliberately by the operation of *arthrodesis*. This may be undertaken to relieve pain or, in tuberculous joints, to prevent recrudescence of the infection.

Decreased mobility, though not complete fixity, may also be caused by fibrous adhesions around a joint without gross intra-articular disease. This condition sometimes results from gonococcal infection.

### A Classification of Diseases of Joints

Injuries of joints due to acute mechanical violence have been described in Chapter VI. The remaining joint disorders form a heterogeneous group which may be classified as follows:

1. Rheumatic fever.
2. Infective arthritis (that is, arthritis due to infection with *demonstrable* micro-organisms).
  - (1) Suppurative arthritis.

- (2) Gonococcal arthritis.
- (3) Tuberculous arthritis.
- (4) Syphilitic arthritis.
3. Rheumatoid arthritis.
4. Osteoarthritis.
5. Neuropathic or 'Charcot's' joints.
6. Gout.
7. Haemophilic joints.
8. Osteochondritis dissecans.

Tuberculous arthritis has been discussed in Chapter XIII; syphilitic arthritis is rare and need not be considered. The remaining conditions, however, require description.

### **Rheumatic Fever**

Rheumatic fever is a disease of medical rather than surgical interest. It is characterized by fever and arthritis, and usually by the development of lesions of the heart and heart valves.

The disease probably results from the patient becoming sensitized to streptococcal toxins. It used to be regarded as a form of infective arthritis, but no organism has been isolated consistently either from the affected joints or from the heart.

The affected joints—most usually the knees and ankles—become red and swollen due to the development of a synovial effusion. Suppuration never occurs. Cardiac disorders and other serious complications may occur in spite of adequate treatment, but the problems they present fall within the province of the physician and need not be discussed here.

All patients should be given a course of penicillin to eliminate haemolytic streptococci from the nose and throat. Aspirin or salicylates and prednisone are helpful in combatting fever and pain, but it is doubtful whether these drugs influence the long term results.

### **Suppurative Arthritis**

Suppurative arthritis may arise in three ways:

1. By blood-borne infection.
2. By spread of infection from a nearby focus; for example, from osteomyelitis in one of the bones taking part in the articulation.
3. By direct infection as a result of a penetrating wound.

The organisms mainly responsible are streptococci, staphylococci, pneumococci and sometimes gonococci. The first two may reach the joint by any of the routes described; the second two are usually blood-borne.

The joint becomes swollen, hot and tender; it is extremely painful if movement is attempted. The synovial membrane is acutely inflamed, the articular cartilage is destroyed and the joint becomes filled with pus. Until recently the mortality was high, and if the patient survived the infection bony ankylosis usually occurred. Nowadays the condition is treated by repeatedly aspirating the pus and replacing it with penicillin solution or by administering some other antibiotic if the organisms are penicillin-resistant. The mortality has been greatly reduced and, if treatment is started early, ankylosis may be prevented, especially in cases due to blood-borne infection.

### Gonococcal Arthritis

The primary lesion in gonorrhoea is situated in the male urethra, the female genital tract, or the eyes of new-born babies. In the male there is acute inflammation in the anterior part of the urethra, and infection may spread to the posterior urethra, prostate, seminal vesicles and testes. The acute inflammation subsides, even without treatment, in a few weeks, but chronic inflammation may persist in the prostate and vesicles for months or years.

Gonococcal arthritis occurs as a result of spread of organisms by the blood stream. Two main types of the disease are seen:

1. *The monarticular type.* The arthritis develops within a few weeks of the original infection. Only one joint is affected, most commonly the knee. Suppuration sometimes occurs, but more often there is a serous or sero-fibrinous effusion into the joint.

2. *The polyarticular type.* The arthritis may develop soon after the original infection; sometimes, however, it appears late, and is then due to spread from a focus of chronic infection, usually in the prostate or one of the seminal vesicles. Many joints are affected, especially the small joints of the hands. The inflammation is non-suppurative and is usually mainly peri-articular. It is often followed by gross limitation of movement due to peri-articular fibrosis.

Penicillin and some of the sulphonamides are effective in the treatment of acute gonococcal infections, but neglected cases in which the infection has become chronic may be difficult to cure.

### Rheumatoid Arthritis

Rheumatoid arthritis is a painful polyarthritis affecting particularly the small joints of the hands (Fig. 93). It is commoner in women than men

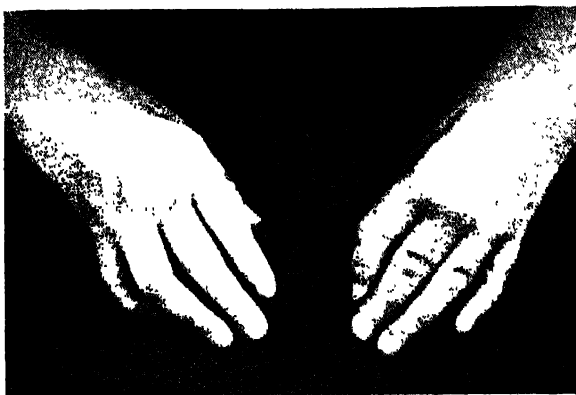


FIG. 93. The hands in rheumatoid arthritis.

and usually begins in middle life. The joint capsule and peri-articular structure are first affected, but later granulation tissue forms in the joint and gradually spreads as a thick vascular sheet (*pannus*) over the articular ends of the bones. Deformity, limitation of movement and sometimes complete fibrous ankylosis ensue, and the patient often becomes seriously crippled.

The etiology is uncertain but there is much evidence to suggest that the condition is a form of autoimmune disease.

Effective treatment depends on the co-ordinated efforts of a team which includes a physician, an orthopaedic surgeon and a physiotherapist. In periods of exacerbation synthetic steroids such as prednisone are of great value, but prolonged administration of these drugs may lead to serious complications including peptic ulcer with associated bleeding or perforation.

In *some* cases removal of a septic focus is followed by improvement in the arthritis; it is, therefore, proper to search for and remove any focus of infection in the mouth, throat or abdomen. On the other hand, it is entirely wrong to remove a patient's teeth, tonsils, appendix, gall bladder, etc., in the absence of definite evidence of infection, simply because he has rheumatoid arthritis.

### **Osteoarthritis**

Osteoarthritis is a degenerative condition involving primarily the articular cartilage. The hip and knee are often affected, but the disease also occurs in the spine and other joints.

In many cases the condition is confined to a single large joint, but this is not invariable. It is common in elderly people, but occurs sometimes in middle age or even in early adult life.

The main etiological factors are:

1. *Senility*.

2. *Recurrent mild trauma*. In the joints of the lower limb, which are subjected to weight bearing, trauma of this kind may result from a mal-united fracture or other deformity which alters the magnitude and direction of the stress transmitted through the joint.

3. *Avascular necrosis* of the articular portion of one of the bones taking part in the joint. This may be caused by a dislocation, or a fracture near the joint, in which blood vessels supplying the end of the bone are torn. Necrosis occurs in the part of the bone deprived of its blood supply, and in the overlying articular cartilage. The dead bone may be slowly replaced by new bone, but the dead cartilage is never replaced and degenerative changes occur in the joint which culminate in the picture of fully developed osteoarthritis.

The main pathological change is erosion of the articular cartilage, especially in its central part. The joint space becomes narrowed, and the bone ends become grooved and *eburnated* (that is, hardened and highly polished). The peripheral part of the cartilage usually escapes erosion, but irregular outgrowths of bone and cartilage appear at this site; these are known respectively as *osteophytes* and *chondrophytes* (Fig. 94).

The symptoms are pain on movement and stiffness after rest. On examination there is palpable and sometimes audible crepitus on movement of the joint. A small synovial effusion may be present. Occasionally an osteophyte or chondrophyte becomes broken off and forms a loose body in the joint (*vide infra*).

The aim of treatment is to reduce the stress applied to the joint by limiting the patient's activity and correcting or compensating for any deformity. If, for example, the arthritis is associated with shortening of one leg, this may be compensated for by raising the heel and sole of the shoe of the affected side.

Physiotherapy, including radiant heat or short-wave diathermy, is of some value. There is no evidence that septic foci play any part in the etiology of the condition and removal of such foci is unlikely to be beneficial. In selected cases pain may be relieved and mobility improved by a plastic operation on the joint known as *arthroplasty*; if this is not feasible and pain is severe the joint may be arthrodesed.





FIG. 94. Radiograph of an osteoarthritic knee. Note the osteophytes on the lateral side of the joint.

### **Neuropathic Joints**

In *tábes dorsalis* and *syringomyelia*—two diseases of the nervous system in which deep sensation is impaired—gross pathological changes may develop in one or more joints. The condition was first described by Charcot and the affected joints are termed *neuropathic* or Charcot's joints (Fig. 48).

At first the lesions resemble those of osteoarthritis; later, as a result of increasing atrophy of cartilage and bone, combined in many cases with hypertrophy around the edges of the articular surfaces and distension due to a synovial effusion, the joint becomes grossly deformed and unstable. Finally, pathological dislocation may occur. Despite these changes the patient experiences little or no pain.

The pathogenesis of Charcot's joints is uncertain. It is possible that owing to the loss of deep sensation the joint suffers repeated injury which passes unnoticed at the time; this alone, however, does not provide an adequate explanation of the condition.

*Treatment.* Suitable apparatus should be provided to increase the stability of the joint and decrease the stress to which it is subjected. In addition, in tabetic cases, appropriate anti-syphilitic treatment must be given.

### Gout

Gout is a disease in which there is a disturbance of purine metabolism. As a result of this sodium urate crystals are deposited in and about joints, especially in the ligaments and articular cartilage; and sometimes also in tendon sheaths and bursae, and in the external ear in the form of nodules known as *tophi*.

The joint most commonly affected is the metatarso-phalangeal joint of the big toe. The patient experiences recurring attacks in which the joint becomes acutely painful and swollen.

Treatment is medical and the disease is of surgical interest only because it may present a problem in differential diagnosis.

### Haemophiliac Joints

Haemophilia has already been mentioned in Chapter III. In this disease bleeding often occurs into joints, causing them to become swollen, hot and painful. Although blood is sometimes completely absorbed from joints repeated bleeding results in clotting, and subsequent organization of the clot leads to limitation of movement or even fibrous ankylosis.

There is no known cure for haemophilia but the patient's life expectancy can be greatly increased by protecting him from injuries and by taking appropriate precautions (Chapter VIII) if any form of surgical operation (including dental extraction) has to be undertaken. If haemarthrosis occurs the joint should be immobilized in the position most favourable for ankylosis.

### Osteochondritis Dissecans

*Osteochondritis dissecans* is a disease of uncertain etiology which occurs in the knee and elbow joint. A piece of articular cartilage, with the subjacent bone to a depth of a few millimetres, becomes gradually separated—'dissected', as it were—from the medial condyle of the femur or the capitellum of the humerus, and set free in the joint. It is then known as a *loose body*.

Loose bodies formed of bone or cartilage may also result from intra-articular fractures, or from detachment of osteophytes or chondrophytes in osteoarthritis. If they remain free in the joint loose bodies give no indication of their presence, but if they come to lie between the articular surfaces of the bones all the symptoms and signs of internal derangement appear. Loose bodies which contain bone are easily recognizable radiologically and should be removed as soon as diagnosed.

### Diseases of the Temporo-Mandibular Joint

Diseases of the temporo-mandibular joint, in contrast to injuries, are comparatively rare. The most important ones are described below.

#### Suppurative arthritis

Suppurative arthritis occurs occasionally. It is usually due to blood-borne infection, but is sometimes caused by direct extension of suppuration from the parotid gland or, in children, from the middle ear.

The joint becomes swollen and painful, movement is limited and the overlying tissues are acutely inflamed.

Aspiration of the pus and replacement with penicillin solution may overcome the infection. Failing this incision and drainage is necessary, but is almost certain to be followed by bony ankylosis. If ankylosis occurs the condyle of the mandible may be excised (Fig. 95). This operation should not be delayed too long, particularly in a child, because lack of use will lead to lack of growth, facial asymmetry and distortion of the developing teeth.

#### Gonococcal arthritis

The temporo-mandibular joint is occasionally involved in gonorrhoea. Suppuration is rare, but peri-articular fibrosis is common. Conservative treatment of the infection with penicillin offers some prospect of retaining a mobile joint, but if there is stiffness due to peri-articular adhesions Esmarch's operation (*vide infra*) may be indicated.



A



B



C



D

FIG. 95. *A and B.* Fixity of the jaw due to ankylosis of the left temporo-mandibular joint. Suppurative arthritis occurred as a complication of acute mastoiditis when the patient was 5 years old.

*C and D.* Restoration of movement following condylectomy.

(Dr Hugh Crombie's case)

**Tuberculosis**

Tuberculosis of the temporo-mandibular joint, though rare, is of some importance because it occurs usually in middle or old age and is easily mistaken for osteoarthritis.

The bone is involved at an early stage of the disease and, if the condition is neglected, sinuses soon develop and secondary infection with pyogenic organisms follows.

Conservative treatment is unsatisfactory as sufficiently prolonged immobilization of the joint is almost impracticable. Excision of the head of the mandible should therefore be undertaken as soon as the diagnosis has been made.

**Osteoarthritis**

Osteoarthritis of the temporo-mandibular joint is fairly common, and is sometimes bilateral. It may be caused by long-standing internal derangement.

The main symptoms and signs are pain, limitation of movement, crepitus, and sometimes visible and palpable irregularity of the condyle of the mandible. The diagnosis is confirmed by X-ray.

Occasionally loose bodies are formed by detachment of osteophytes, as in osteoarthritis elsewhere; and in a few cases degenerative changes in the joint progress to such a stage that partial pathological dislocation occurs.

If the symptoms are severe the only effective treatment is excision of the condyle of the mandible.

**Limitation of movement of the mandible**

Limitation of movement of the mandible may develop suddenly (*acute limitation*) or gradually (*chronic limitation*). In either case the cause may lie in the temporo-mandibular joint (*intrinsic causes*) or in structures outside the joint (*extrinsic causes*).

*Acute limitation*

1. *Intrinsic causes.* There are three lesions of the temporo-mandibular joint which may cause acute limitation of movement of the mandible:

- (1) Traumatic synovitis (Chapter VII).
- (2) Internal derangement (Chapter VII).
- (3) Suppurative arthritis.

2. *Extrinsic causes.* Acute limitation of movement of the mandible due to extrinsic causes is known as *trismus*. It is caused by spasm of the muscles

of mastication, particularly the masseter and the medial pterygoid, and may occur in the following conditions:

- (1) Tetanus.
- (2) Strychnine poisoning.
- (3) Painful inflammatory conditions in the vicinity of the temporo-mandibular joint. These include: mumps, suppurative parotitis, acute lymphadenitis, acute tonsillitis, an impacted and infected third molar tooth, alveolar abscess and osteomyelitis of molar origin.
- (4) Facial fracture.
- (5) Hysteria.

### *Chronic limitation*

1. *Intrinsic causes.* The lesions of the temporo-mandibular joint which cause chronic limitation of movement of the jaw are as follows:

- (1) Fibrous or bony ankylosis following infective arthritis.
- (2) Internal derangement.
- (3) Osteoarthritis.

2. *Extrinsic causes.* These are as follows:

- (1) Cicatricial contracture of the peri-articular tissues due to gonococcal arthritis, burns, lupus, actinomycosis, operations, radiotherapy, etc.
- (2) Excessive callus formation following fracture of the neck of the mandible. \*This is a rare condition.
- (3) Malignant neoplasms of the face or mouth.

### *Treatment*

The first essential is to maintain nutrition. In chronic cases the patient has usually learned to feed himself adequately, but in acute cases tube-feeding may be necessary.

Further treatment depends on the underlying cause. The treatment of most of the conditions enumerated above has already been described but limitation of movement due to cicatricial contracture of the peri-articular tissues remains to be considered. This condition cannot be relieved by condylectomy. In mild cases mobility can be restored by excising the scar tissue, but sometimes it is necessary to create a false joint by removing a wedge of bone, with its apex at the alveolar border, from the angle of the mandible. This is known as Esmarch's operation.

## *Chapter XXI*

# SOME SURGICAL DISORDERS OF THE NERVOUS SYSTEM

In discussing disorders of any system of the body a distinction is often drawn between *organic* disorders and *functional* disorders; this distinction is of especial importance when dealing with the nervous system.

An organic disorder is defined as one in which there is some demonstrable abnormality of structure; a functional disorder as one in which there is an abnormality of function but no demonstrable structural abnormality.

The distinction, though important, is by no means absolute, since it seems probable that in some apparently functional disorders such as trigeminal neuralgia the development of more refined methods of histological examination will lead to the discovery of structural abnormalities which at present elude detection. On the other hand most neurologists, whatever their views on the vexed question of the relationship between mind and body, would agree that there are some conditions (for example, so-called *hysterical paralysis*) which are essentially mental and for which no structural basis is ever likely to be discovered.

Many nervous disorders fall within the province of the physician; many of the remainder, which are of surgical importance, are beyond the scope of this book. The dental student should, however, understand the principles underlying the diagnosis of lesions of the spinal cord and cranial nerves, and should have a detailed knowledge of trigeminal neuralgia.

### **The Spinal Cord and the Peripheral Spinal Nerves**

A pair of nerves arises from each segment of the spinal cord. Each nerve is connected to the cord by two roots, an anterior root carrying *efferent* or motor fibres and a posterior carrying *afferent* or sensory fibres. Connected to each posterior root is a ganglion, the posterior root ganglion.

Some of the spinal nerves join together to form plexuses (for example, the brachial plexus) from which peripheral nerve trunks arise. These trunks may contain fibres derived from several segments of the cord. Most peripheral nerve trunks and their larger branches contain both motor and sensory fibres and are termed mixed nerves; a few are purely motor or purely sensory.

*The motor pathway*

Motor impulses arise in the large pyramidal cells of the motor area of the cerebral cortex. The axons of these cells descend to the brain stem; most of them then cross over to the opposite side and continue in the spinal cord where they constitute the 'crossed pyramidal tracts'. They end by forming synapses with the motor cells in the anterior horns of the grey matter of the spinal cord. The axons of the anterior horn cells leave the cord in the anterior spinal nerve roots, and travel in the spinal nerves and their branches to the voluntary muscles (Fig. 96).

Two nerve cells or neurones are thus concerned in the passage of an impulse from the motor cortex to a voluntary muscle: one, known as the *upper motor neurone*, situated in the cortex; the other, known as the *lower motor neurone*, situated in the spinal cord.

Damage to motor neurones causes paralysis of the corresponding muscles. In upper motor neurone paralysis, although voluntary movement is lost, the affected muscles remain healthy and still contract as a result of reflex action; in lower motor neurone paralysis, on the other hand, the muscles lose all power of contraction and begin to waste after a few weeks.

Other parts of the brain besides the motor cortex send connections to the anterior horn cells of the cord and thus play a part in the regulation of voluntary movement, but the simple picture described above is accurate as far as it goes and adequate for our present purpose.

*The sensory pathway*

Receptor organs of various types exist in the skin, muscles, tendons and other parts of the body.

The neurones of the posterior root ganglia send processes peripherally via the spinal nerves to the receptors in the skin, muscles and other structures, and centrally to the spinal cord. Of these central fibres the ones carrying sensations of pain and temperature, and some of these concerned with tactile sensation, form synapses with cells in the posterior horns of the grey matter of the cord; the axons from these cells cross to the opposite side of the cord and ascend in the spino-thalamic tracts to the thalamus. The fibres conveying sensory impulses from muscles and tendons, which give information about movement and the position of joints, and the remainder of the fibres concerned with tactile sensation, ascend in the posterior column on the same side of the cord to nuclei in the medulla oblongata; here they form synapses with cells whose axons ascend to the thalamus.

From the thalamus a third relay of fibres ascends to the sensory area of the cerebral cortex (Fig. 97).



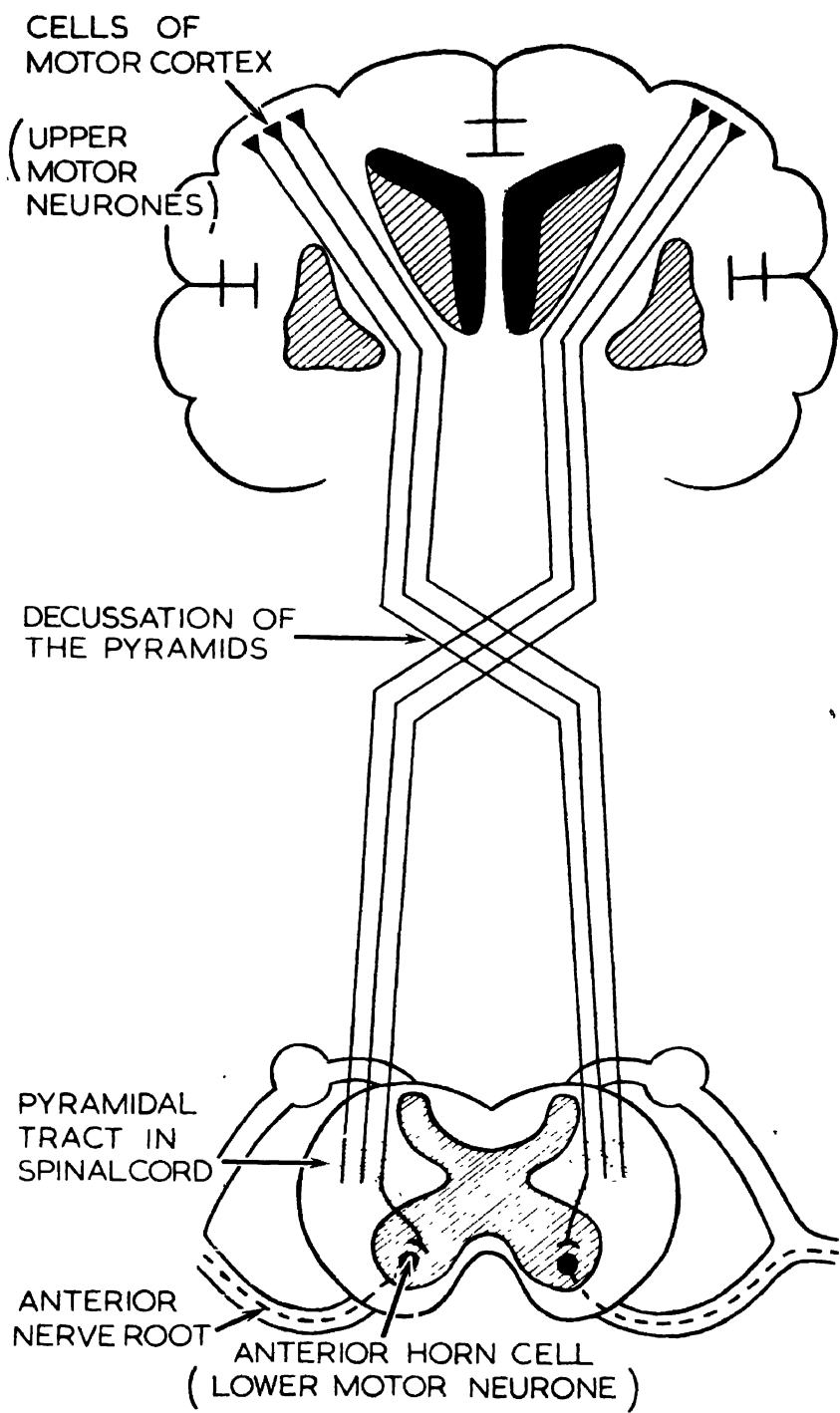


FIG. 96. The motor pathway.

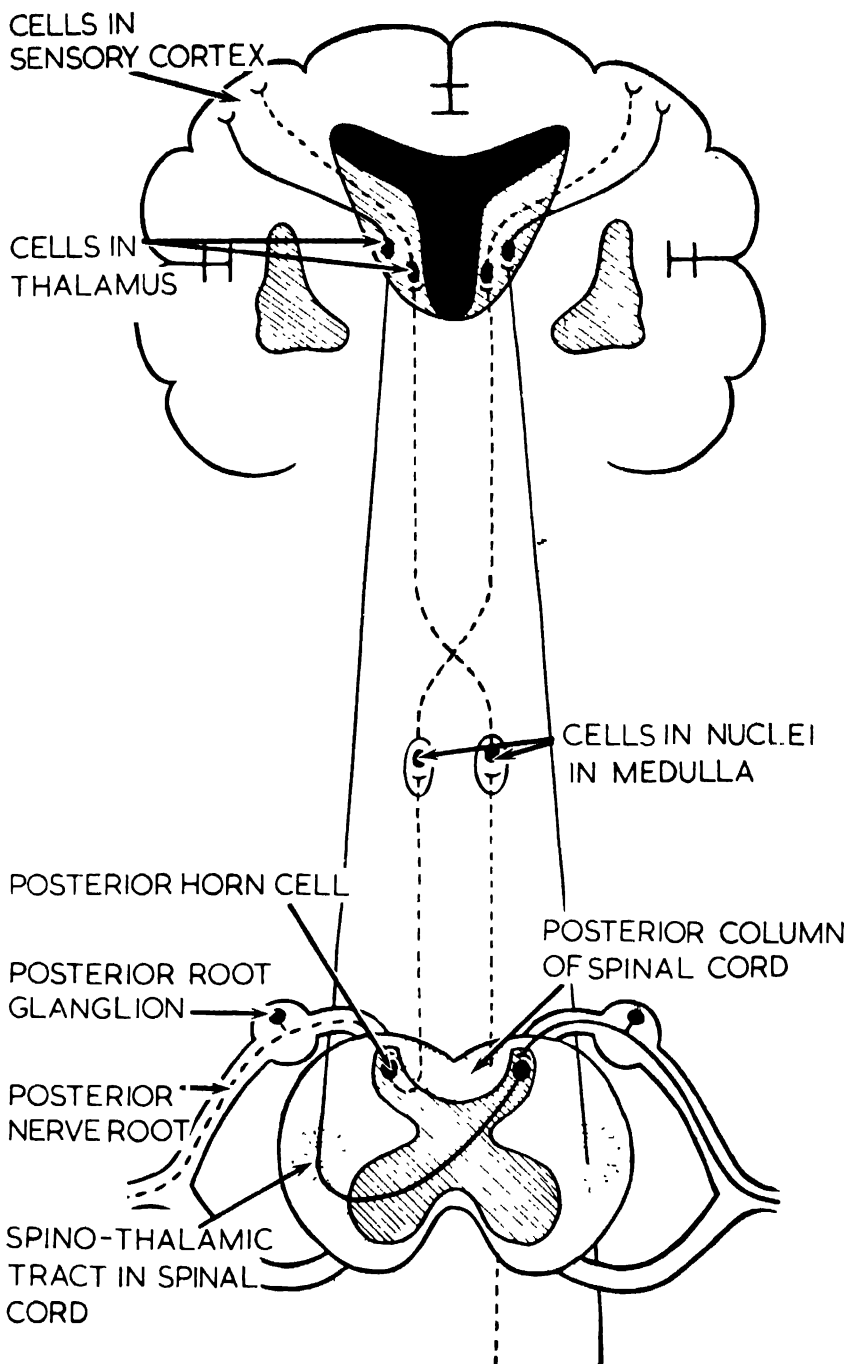


FIG. 97. The sensory pathway.

### Injuries of the spinal cord

The common causes of lesions of the spinal cord met with in surgical practice are injuries of the spine (fractures and dislocations), tuberculosis of the spine and spinal tumours.

In *injuries of the spine* the passage of impulses may be temporarily prevented without gross injury to the cord; this is called spinal concussion. Alternatively, the cord may be contused or actually severed. In spinal tuberculosis the cord may be compressed by a cold abscess, tuberculous granulation tissue or a displaced piece of bone; alternatively, it may be deprived of its blood supply by thrombosis in the vessels. *Tumours* may develop within the cord (intramedullary tumours) or grow in the spinal canal (extramedullary tumours) and compress the cord.

If fibres in the cord are divided the effects are permanent; fibres which have been compressed may recover their function if the pressure is relieved. A lesion is described as *complete* if the transmission of impulses is totally and permanently abolished; if some fibres continue to transmit impulses or recover their function after a time the lesion is called *incomplete*.

The effects depend on the degree of damage to the cord and the level of the lesion.

In complete lesions there is paralysis of the upper motor neurone type and complete anaesthesia below the level of the lesion; there are also disturbances of micturition and defaecation. If the injury is above the level of the fourth cervical segment of the cord all the muscles of respiration are paralysed and death occurs rapidly from asphyxia unless the patient is maintained by mechanical artificial respiration. With lower cervical lesions the diaphragm escapes but the intercostal muscles are all paralysed; the patient survives for a time, but usually dies from pneumonia within a week or two. With lesions in the mid-dorsal region, the upper intercostal muscles retain their function; respiration is adequate but the patient is totally paralysed and anaesthetic from the waist downwards. Bedsores frequently develop and the patient is unable to empty his bladder; after a time the bladder may empty automatically when it is full, but voluntary control of micturition is not regained.

Incomplete lesions are more common than complete ones; the effects depend on which fibres are affected. With spinal tumours the damage to the cord steadily increases and there is sometimes a stage when one-half of the cord is functioning and the other is not. There is then upper motor neurone paralysis on the same side of the body as the lesion, and loss of the sensations of pain and temperature on the other side. For example, with a tumour pressing on the right half of the lumbar part of the cord

there may be paralysis of the right lower limb and anaesthesia of the left one.

### **Peripheral nerve injuries**

A peripheral nerve may be completely or partially divided in a wound, or it may cease to function, without being divided, as a result of stretching, contusion or compression.

*Stretching* may occur as a complication of a dislocation. Usually nerve fibres are torn but the sheath remains intact. *Contusion* may be caused by a blow, or by a bullet passing close to the nerve.

The causes of *compression* of a nerve may be grouped under four headings, as follows:

1. Pressure from outside the body; for example, the pressure of a crutch in the axilla or a badly applied plaster.
2. Bony malformations; for example, a cervical rib.
3. Involvement of a nerve in scar tissue or in the callus of a healing fracture.
4. Tumours and aneurysms. An important example is compression of the facial nerve by a carcinoma of the parotid gland.

Division of a motor nerve causes paralysis of the lower motor neurone type in the muscles it supplies.

Division of a sensory nerve causes anaesthesia. There is usually an area in which all sensation is lost, surrounded by an area in which crude sensations such as severe pain and gross changes in temperature are appreciated, but fine sensations such as the appreciation of light touch are lost.

Division of a mixed nerve results in both paralysis and anaesthesia.

When a nerve is divided its distal part undergoes degeneration. New fibres grow out from the proximal part and, if the nerve ends are brought into apposition, enter the distal part and grow along the path of the degenerated fibres. Even in favourable cases recovery is slow because the fibres from the proximal part of the nerve grow only about 1 mm. per day; in time, however, function may be largely restored after division of a purely sensory or purely motor nerve. With mixed nerves the results are much less satisfactory; this is because, however accurately the nerve ends are opposed, a certain number of motor fibres make their way along the track of degenerated sensory fibres, and vice versa.

After *partial* division of a sensory or mixed nerve, notably the median nerve at the wrist, severe and very intractable pain sometimes develops. This condition is known as *causalgia*.

*Treatment*

If a nerve has been completely divided in a clean wound it should be repaired immediately. The ends are brought into accurate apposition without rotational deformity and joined together by sutures passed through the nerve sheath. If the wound is contaminated nerve suture is postponed until infection has subsided.

After nerve suture, and before operation if suture has to be delayed, splints are applied to protect paralysed muscles from overstretching, and anaesthetic areas are protected from injury. Joints which do not have to be immobilized are exercised daily, and massage and electrical stimulation are used to maintain tone in the paralysed muscles. Later, when signs of recovery appear, special exercises are prescribed to re-educate the patient in the use of muscles which have been paralysed.

Sometimes it is impossible to tell whether a nerve has been divided or merely contused. In these cases the same precautions are taken to protect paralysed muscles and anaesthetic areas. After 4 to 6 weeks, if there is no evidence of returning function, the nerve is explored and freed from scar tissue; if it is found to be divided it is sutured as described above.

### **The Cranial Nerves**

*The olfactory (first cranial) nerve*

The olfactory nerve is sometimes injured in fractures of the base of the skull. The only clinical sign of injury is loss of the sense of smell (*anosmia*) on the affected side.

*The optic (second cranial) nerve*

Injuries of the optic nerve proper are rare, but a degenerative process termed *optic atrophy* occurs in a number of diseases of the nervous system. Complete destruction of the nerve results in total blindness in the corresponding eye. Pressure on the optic chiasma by a tumour of the pituitary gland causes defects in the visual fields which progress to blindness unless the tumour is removed.

*The oculomotor, trochlear and abducent (third, fourth and sixth cranial) nerves*

These nerves, which control movements of the eyeball, are sometimes damaged in fractures of the base of the skull, and sometimes compressed by intracranial haemorrhage or tumours.

Lesions of the oculomotor nerve result in *ptosis* (that is, drooping of the upper lid), outward deviation of the eye and dilatation of the pupil on the affected side; *diplopia* (that is, double vision) occurs because the visual axes are not parallel.

Lesions of the trochlear nerve cause paralysis of one muscle only, the superior oblique. The patient complains of diplopia, especially when looking downwards and outwards, but there is no noticeable deviation of the visual axes.

Lesions of the abducent nerve causes paralysis of the external rectus muscle. If the patient is asked to look towards the side of the lesion the sound eye moves but the affected eye remains looking straight ahead.

#### *The trigeminal (fifth cranial) nerve*

It is essential for a dental student to have a detailed knowledge of the anatomy of the trigeminal nerve, and it is assumed that the reader possesses this knowledge.

The trigeminal nerve is sometimes injured in fractures of the base of the skull and sometimes damaged by compression. Division of the sensory root of the Gasserian ganglion, which results in anaesthesia in the area of distribution of the whole nerve, is sometimes undertaken for the treatment of trigeminal neuralgia (*vide infra*).

The ophthalmic and maxillary branches of the nerve contain only sensory fibres, and lesions of these branches cause anaesthesia in the areas which they normally supply (Fig. 98). The territory of the ophthalmic branch includes the cornea; anaesthesia of this structure is often followed by ulceration and sometimes leads to complete disorganization of the eye and blindness.

A lesion of the mandibular branch of the nerve results in anaesthesia in the lowest of the three areas shown in Figure 98, including the lower jaw and the anterior two-thirds of the tongue; it also causes lower motor neurone paralysis of the muscles of mastication. This latter phenomenon can best be detected by palpating the masseter muscle while the patient attempts to clench his teeth.

#### *The facial (seventh cranial) nerve*

The facial nerve is a purely motor nerve which supplies the muscles of facial expression. The nerve trunk may be injured or compressed within the skull, in its course through the petrous bone, or in the substance of the parotid gland. The branches, which arise from the trunk of the nerve in the substance of the parotid, are sometimes injured in wounds of the face and in operations on parotid tumours.

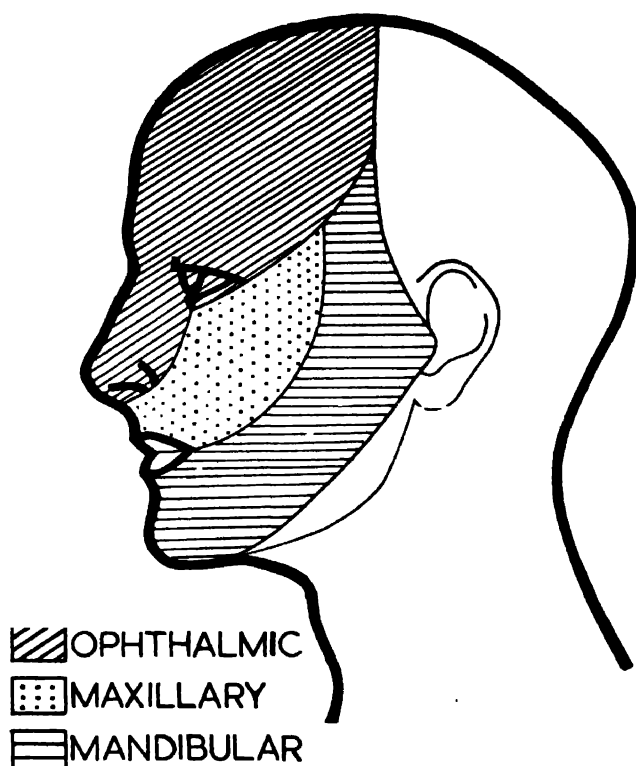


FIG. 98. Sensory distribution of the trigeminal nerve.



FIG. 99. Complete facial paralysis. The patient has been asked to smile and screw up his eyes at the same time. On his left side movements are normal; on his right side all the muscles supplied by the facial nerve are paralysed.

Facial paralysis may also develop in the absence of injury or any gross lesion inside the skull, probably as the result of a virus infection. This condition, which is fairly common, is known as *Bell's palsy*.

The diagnosis of complete facial paralysis is obvious on inspection (Fig. 99). The affected side of the face is expressionless and immobile; the mouth is deviated towards the normal side. If the patient is asked to wrinkle his forehead, whistle or show his teeth, there is no movement on the affected side. If he tries to close his eyes the eye on the affected side looks upward but the lids do not close.

If one of the branches of the facial nerve is injured there is paralysis of *part* of the face on the side of injury.

Sometimes the facial nerve is intact, but the fibres running from the motor cortex to the nucleus of the nerve in the brain stem are damaged by cerebral haemorrhage or by a brain tumour. In these cases, if the lesion is unilateral, there is paralysis of the lower half of the face on the side opposite to the lesion in the brain, but the upper part of the face is not paralysed. This is because the muscles of the lower part of the face are controlled by the motor area on the opposite side of the brain, whereas each muscle in the upper part of the face is controlled by the motor areas of *both* sides. To cause paralysis of the upper part of the face a brain lesion must therefore be bilateral.

Facial paralysis is disfiguring. When the paralysis is likely to be temporary, as in Bell's palsy, it is desirable to protect the muscles from overstretching. The usual procedure is to use a hook in the angle of the mouth connected by a fine wire to a larger hook encircling the ear on the same side; it is better, however, to apply strips of adhesive strapping, and extend these across to the sound side of the face so as to overcome the pull of the non-paralysed muscles. When the nerve has been divided function can sometimes be restored by suturing the nerve or by a nerve graft; alternatively, the patient's appearance may be improved by a plastic operation.

#### *The auditory (eighth cranial) nerve*

The auditory nerve is the nerve of hearing; it also supplies the vestibule of the ear, which is concerned with balancing.

The nerve may be compressed by a tumour arising from the nerve sheath or injured in fracture of the base of the skull. Damage to the auditory part of the nerve results in deafness on the affected side.

#### *The glossopharyngeal (ninth cranial) nerve*

The glossopharyngeal nerve is the sensory nerve to the pharynx and posterior third of the tongue. It is rarely injured.



*The vagus (tenth cranial) nerve*

The vagus nerve carries parasympathetic fibres to the heart, the lungs and much of the alimentary tract. In addition, it gives branches to the muscles of the pharynx, and two branches to the larynx which between them constitute the whole motor and sensory supply of that structure.

The main trunk of the vagus is rarely injured, but the lower of the two branches to the larynx, the recurrent laryngeal nerve, is sometimes compressed by tumours of the thyroid gland or injured during the operation of thyroidectomy. The symptoms which result include hoarseness and some difficulty in breathing. The diagnosis is confirmed by observing the vocal cords through a laryngoscope while the patient is asked to take a deep breath; the cord on the side of injury remains stationary close to the mid-line while the opposite cord moves outwards. If both recurrent laryngeal nerves are injured the patient has such difficulty in breathing that tracheostomy may be necessary.

*The spinal accessory (eleventh cranial) nerve*

The spinal accessory nerve joins with the vagus in supplying the muscles of the pharynx. It also supplies the trapezius muscle and assists in supplying the sternomastoid.

The nerve is sometimes injured during operations on the neck; for example, in removal of tuberculous glands. The resulting paralysis of the trapezius muscle becomes apparent if the patient is asked to shrug his shoulders.

*The hypoglossal (twelfth cranial) nerve*

The hypoglossal is the motor nerve to the tongue. If it is injured the tongue deviates to the affected side when protruded, and after some weeks the muscles of the tongue on the same side become wasted.

**Trigeminal Neuralgia (*Tic Douloureux*)**

Trigeminal neuralgia has been defined by Trotter as a 'chronic progressive disease of which the sole primary symptom is pain in the distribution of the fifth cranial nerve. There is no impairment of sensation or of the motor function of the nerve, and no causal relationship to peripheral foci of infection.'

The disease usually appears in middle life and is equally common in men and women. At first the pain is limited to one division of the nerve, usually the maxillary or mandibular. There is often hyperaesthesia in the

region in which the pain occurs. In many patients 'trigger areas' exist; stimulation of one of these, even by light touch, may precipitate a bout of pain.

When the maxillary division is involved the pain usually begins in the upper lip and spreads to the teeth, palate, lower eyelid and cheek. When the mandibular division is involved the pain occurs in the lower lip and teeth, the anterior two-thirds of the tongue, the temporal region and the external auditory meatus.

The patient experiences *attacks*, lasting from a week to several months, during which there is continual discomfort. Between attacks the patient is entirely free from pain. At intervals during an attack violent *bouts* of pain occur, either spontaneously or following stimulation of a trigger area. The bouts last from a few minutes to an hour or more; during this time the patient remains immobile except sometimes for occasional chewing movements, his face is flushed and his eyes water.

The general health remains unaffected except in so far as some patients become suicidal or develop a drug addiction owing to the severity of the pain.

No morphological changes in the trigeminal nerve, the Gasserian ganglion or the brain have been demonstrated.

## **Diagnosis**

The diagnosis is made on the history of bouts of severe pain in the distribution of the trigeminal nerve, the occurrence of intervals of complete freedom from pain, the absence of any anaesthesia or muscular paralysis, and the lack of any obvious cause.

The conditions which have to be distinguished from trigeminal neuralgia may be classified as follows:

### *1. Pains of trigeminal distribution*

#### *(1) Neuralgic pain associated with a peripheral focus of infection.*

This is a common condition, and the differential diagnosis from true trigeminal neuralgia is important because the treatment is entirely different.

The lesions mainly responsible are sinusitis, an impacted wisdom tooth and sclerosis round a tooth socket following infection.

The pain rarely occurs in definite attacks and bouts, and is usually not rigidly restricted to the distribution of a particular division (or divisions) of the trigeminal nerve.

The treatment of this condition is, of course, to deal with the underlying cause.

*(2) Neuralgic pain following herpes zoster (shingles).*

Herpes zoster is characterized by severe pain along the course of one or more nerves, together with inflammation and vesicle formation in the areas of skin which they supply. The disease is due to a virus infection of the posterior root ganglia of the spinal nerves or, less commonly, the Gasserian ganglion. When the Gasserian ganglion is affected the pain and skin changes occur in the distribution of the ophthalmic division of the trigeminal nerve and the cornea of the eye may be affected. Usually the pain appears a few days before the rash and both disappear after a week or two; occasionally the pain persists for months. Persistent pain due to ophthalmic herpes is distinguished from trigeminal neuralgia by the history of typical herpetic lesions in the skin and the cornea.

*(3) Neuralgic pain due to an organic lesion involving or pressing on the nerve trunk or its branches.*

This condition is not very common, and it is unnecessary for our present purpose to attempt to classify the possible causal lesions. The differentiation from true trigeminal neuralgia is not difficult because there is nearly always some anaesthesia, or loss of power in the muscles of mastication.

*2. Pains of distribution other than trigeminal*

Pain resembling that of trigeminal neuralgia, except in its distribution, sometimes occurs in the territory of other nerves, especially the glossopharyngeal. The differential diagnosis depends on the patient's own description of the distribution of the pain.

**Treatment**

Many methods of treatment have been tried in trigeminal neuralgia. The following are in use to-day:

1. *Injection of alcohol into one of the main divisions of the nerve.* This gives only temporary relief; perhaps 18 months in favourable cases.

2. *Injection of alcohol into the Gasserian ganglion.* This also gives relief which is only temporary though it lasts longer than that given by the previous method. The procedure is somewhat dangerous because if alcohol is accidentally introduced into the subarachnoid space it may cause paralysis of the oculomotor, trochlear, abducent and facial nerves.

3. *Resection of the sensory root of the Gasserian ganglion.* This is a difficult operation but carries a low mortality in the hands of an experienced neurosurgeon. Complete division produces anaesthesia in the whole distribution of the nerve, including the cornea, and may be followed by corneal ulceration. Nowadays, therefore, many surgeons divide only about two-thirds

of the fibres of the root and in this way preserve corneal sensation. In all cases pain is relieved at the cost of producing extensive anaesthesia, and a few patients find the anaesthesia almost as distressing as the pain which preceded it.

4. *Division of the descending root of the trigeminal nerve in the medulla.* This procedure is said to produce freedom from pain without causing complete anaesthesia. The operation is difficult and dangerous, but in highly-skilled hands is effective in selected cases.

## Chapter XXII

# DISORDERS OF THE ENDOCRINE GLANDS

Disorders of endocrine glands are commonly associated with excessive or deficient production of the normal glandular secretions (*hormones*).

Excessive hormone production may result from hyperplasia or from neoplastic disease. Hyperplasia may be localized to one or more areas or may involve the whole gland. Neoplasms of endocrine glands may be benign or malignant; excessive hormone production may occur in either case but is more common with benign tumours.

Deficient production of hormone may be due to one of several causes. These include congenital maldevelopment or operative removal of an endocrine gland, inhibition of secretion by drugs, lack of hormonal stimulus normally supplied by some other gland, and destruction of secretory tissue as a result of inflammation or tumour growth.

Enlargement of an endocrine gland is often associated with excessive secretion, but may also occur when secretion is normal or deficient. It may result in pressure on important structures in the neighbourhood.

### Principles of treatment

#### *Disorders due to excessive production of hormone*

Disorders due to excessive production of hormone, such as hyperthyroidism, may sometimes be successfully treated medically, but surgery is usually the method of choice. Irradiation methods have also been used, but are of limited value.

Operations may take various forms; namely:

1. Excision of an adenoma or localized area of hyperplasia; for example, excision of a thyroid adenoma.
2. Subtotal excision of a hyperplastic gland, leaving only a small portion sufficient to meet the ordinary needs of the body. A common example is subtotal thyroidectomy for hyperthyroidism.
3. Complete excision of a hyperplastic gland or a gland containing a tumour. A hyperplastic suprarenal, for example, may be excised for the treatment of *virilism* (*vide infra*).

*Disorders due to deficient production of hormone*

Treatment of disorders due to deficient production of hormone is usually medical and consists in giving the appropriate hormone. Oral administration is sometimes successful, notably in the treatment of myxoedema (*vide infra*); otherwise the hormone is given by injection or in the form of a pellet implanted in the sheath of the *rectus abdominis* muscle.

Attempts have been made to treat endocrine deficiencies by transplantation of the appropriate tissue from another individual or even from an animal, but so far with little success.

*Other indications for treatment*

Apart from the treatment of anomalies of secretion, operation may be undertaken to remove a malignant tumour or to relieve pressure on neighbouring structures caused by benign enlargement of an endocrine gland. The removal of pituitary tumours, for example, is undertaken primarily to relieve pressure on the optic chiasma and the brain; and removal of a colloid goitre (*vide infra*) may be necessary to relieve dyspnoea due to pressure on the trachea.

**The Pituitary Gland**

Increased secretion may be due to an adenoma of the eosinophil or basophil cells of the anterior lobe of the pituitary. Other pituitary tumours, by exerting pressure on the surrounding secretory cells, may cause diminished secretion (*pituitary deficiency*). Pituitary deficiency also occurs when secretory cells are destroyed as a result of haemorrhage or thrombosis.

An eosinophil adenoma results in gigantism or acromegaly (Chapter XIX). A basophil adenoma is sometimes present in patients with Cushing's syndrome, which is characterized by adiposity, hirsutes, hypertension and cyanosis. The clinical features of pituitary deficiency are described in textbooks of medicine.

*Treatment of pituitary tumours*

Correction of the effects of disordered secretion is rarely possible, but surgical removal of the tumour is often required to relieve pressure on the optic chiasma and on the brain. Pressure on the chiasma, if long continued, causes blindness.

## The Thyroid Gland

### Goitre

The term *goitre* is sometimes used to denote any enlargement of the thyroid gland; more commonly, however, it is restricted to enlargement due to causes other than inflammation or neoplasia. We shall use the term in the latter sense.

Enlargement of the thyroid may be due to the development of one or more nodules in the gland, or to a diffuse process involving the whole gland. In either case there may, or may not, be evidence of hyperthyroidism (excessive secretion). Hyperthyroidism is sometimes called 'thyrotoxicosis' and a goitre which produces it is called a 'toxic goitre'. We thus arrive at the following simple classification of goitre:

1. Diffuse goitre:
  - (1) Non-toxic—often called 'colloid goitre'.
  - (2) Toxic—often called 'primary thyrotoxicosis', 'primary Graves' disease' or 'exophthalmic goitre'.
2. Nodular goitre:
  - (1) Non-toxic.
  - (2) Toxic—often called 'secondary thyrotoxicosis' or 'secondary Graves' disease'.

### Diffuse non-toxic goitre

Diffuse non-toxic goitre may be further subdivided into simple colloid goitre and lymphadenoid goitre.

Colloid goitre (Fig. 100) is sometimes *endemic*—that is, it occurs particularly in certain areas, such as the Peak District in Derbyshire, where the soil is deficient in iodine; it also occurs *sporadically*. Apart from the thyroid enlargement there may be no symptoms, but sometimes dyspnoea or dysphagia develops due to the pressure exerted by the enlarged gland. In the absence of pressure symptoms surgery is not usually required, but is sometimes undertaken for cosmetic reasons. If pressure symptoms occur subtotal thyroidectomy should always be performed. Myxoedema may develop after the operation, but responds readily to medical treatment.

Lymphadenoid goitre (often referred to as *Hashimoto's disease*) is a form of autoimmune disease in which there is uniform enlargement of the thyroid gland, associated with infiltration of plasma cells and lymphocytes and destruction of secretory tissue leading to hypothyroidism (*vide infra*). Serological studies reveal the presence of antibodies to antigens of thyroid origin including, as a rule, the protein thyroglobin.

Administration of thyroxine corrects the hypothyroidism and usually causes the gland to become smaller. Operation is not ordinarily required but is undertaken occasionally if there are severe pressure symptoms or if the diagnosis is uncertain.

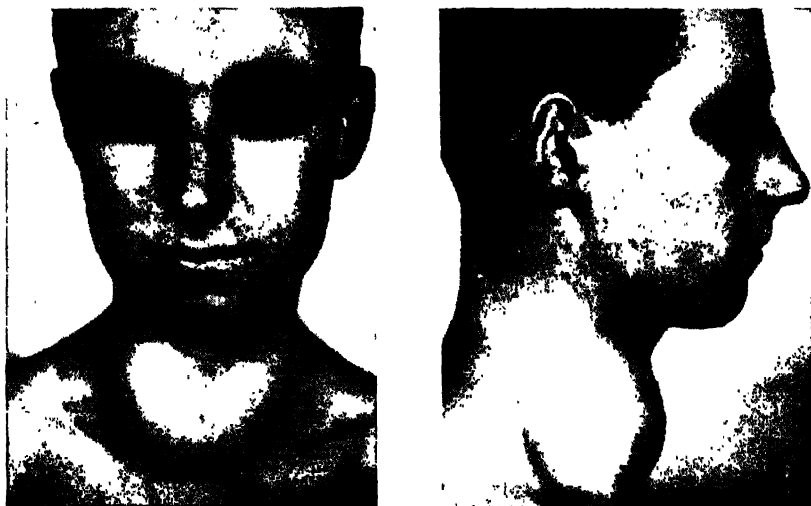


FIG. 100. Diffuse non-toxic goitre.

### Diffuse toxic goitre

Diffuse toxic goitre may occur in either sex, but is much commoner in women. It is rare in childhood but common in early adult life. The condition usually develops suddenly in a patient who has had no previous thyroid disorder; hence the name *primary* thyrotoxicosis. A severe fright or shock sometimes appears to precipitate the onset of the disease, but apart from this fact little is known about its etiology.

#### *Symptoms and signs*

In a typical case there is slight or moderate diffuse enlargement of the thyroid, together with evidence of toxicity and exophthalmos.

The toxic manifestations include nervousness, tremor, dislike of hot weather, loss of weight despite a good appetite, and increased pulse rate. The pulse rate taken when the patient is asleep is of special significance because tachycardia of nervous origin does not persist during sleep whereas that due to thyrotoxicosis does. Cardiac irregularity, including auricular fibrillation, sometimes occurs but is less common than in secondary thyrotoxicosis.



Exophthalmos (Fig. 101) is usually obvious on inspection, but in early cases certain associated signs may be helpful. These include lagging of the upper lid when the patient is asked to look downwards, absence of furrowing of the forehead when he looks upwards, and difficulty in convergence when he tries to look at near objects. Various names are given to these signs, but it is pointless to burden the memory with them.

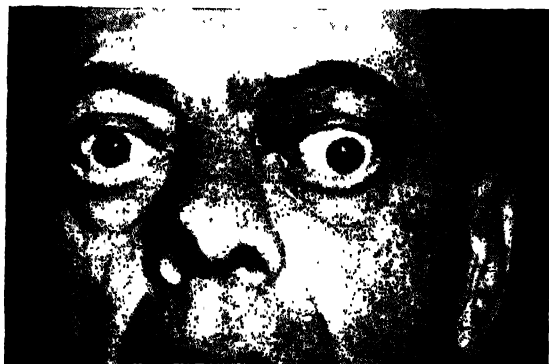


FIG. 101. Exophthalmos in a patient with diffuse toxic goitre. There is also a cataract in the left eye.

Special tests which are useful in estimating the degree of toxicity include estimation of the level of protein-bound iodine in the patient's serum and the uptake of radioactive iodine. Since these tests were developed estimation of the patient's basal metabolic rate, which used to be routine, has ceased to be undertaken in most clinics.

### *Treatment*

Patients should be treated medically in the first instance with antithyroid drugs such as carbimazole and methyl-thiouracil.

Once the patient has been made *euthyroid* (i.e. the level of thyroid function has been brought back to normal) the choice lies between (a) continuing with a small maintenance dose for a year or more and then trying to wean the patient off the drug; (b) performing subtotal thyroidectomy; and (c) destroying the gland by treating the patient with radioactive iodine.

Medical treatment with antithyroid drugs alone gives excellent results in some patients, but many patients relapse and a few develop toxic complications of which the most serious is agranulocytosis (Chapter XI).

Surgical treatment saves much time; in competent hands, and with proper pre-operative and post-operative treatment, the results are excellent and the operative mortality is less than 1 per cent.

Before operation the patient is given a course of Lugol's solution (iodine and potassium iodide) for 2 to 3 weeks; if there are severe toxic symptoms this is preceded by a period of treatment with thiouracil.

The operation is called 'subtotal thyroidectomy' because a small portion of each lateral lobe is left behind. The object of this is to avoid injury to the recurrent laryngeal nerves and to ensure that some parathyroid tissue is preserved; it also reduces the chance of myxoedema developing subsequently.

Administration of radioactive iodine is usually reserved for patients over the age of 45, and is especially indicated when, as occasionally happens, the patient has relapsed after operation. It is completely contraindicated during pregnancy.

Many patients after treatment with radioactive iodine become hypothyroid, but can easily be controlled with thyroxine provided that they remain under proper supervision. There is in theory a risk that treatment with radioactive iodine will lead to the development of neoplastic disease, but in practice this appears to be very small.

Special measures may be required for the treatment of progressive exophthalmos, but these are beyond the scope of this book.

### **Nodular goitre**

Nodular goitre begins as a non-toxic condition, but sooner or later toxic symptoms may develop.

The nodules comprising the goitre may be solid or cystic. Dyspnoea, due to compression of the trachea, is common; it may develop gradually as the goitre enlarges or suddenly owing to haemorrhage into a cyst.

When the goitre becomes toxic there is a rapid pulse, an increased basal metabolic rate, dislike of hot weather, and loss of weight despite a good appetite. Cardiac manifestations, including auricular fibrillation and in some cases cardiac failure, are particularly liable to supervene, but exophthalmos is unusual.

Occasionally carcinomatous change develops in a nodular goitre.

### *Treatment*

In view of the likelihood of pressure symptoms or severe cardiac damage developing, and the slight but definite risk of malignant change, operative treatment should be undertaken for every nodular goitre. If there is no evidence of toxicity and only a single nodule is present this nodule alone

may be removed; otherwise subtotal thyroidectomy should be performed. In toxic cases pre-operative preparation with antithyroid drugs and iodine is required and digitalis may be given if auricular fibrillation is present. If fibrillation persists after operation normal rhythm may often be restored by giving quinidine.

### **Neoplasms of the thyroid gland**

#### *Adenoma*

The nodules which occur in a nodular goitre are often loosely termed adenomata. A true benign tumour, called a 'foetal adenoma' because of its histological resemblance to foetal thyroid tissue, develops occasionally in a normal gland or in one which is already the site of a goitre; it is peculiarly liable to undergo malignant change. It is impossible to distinguish clinically between a foetal adenoma and a solitary goitrous nodule; this is an additional reason for advising surgical treatment in every case of nodular thyroid enlargement.

#### *Carcinoma*

Carcinoma of the thyroid is more common in women than men; it usually occurs after the age of 50, but is seen occasionally in younger patients.

The tumour may develop in a previously normal thyroid, but more commonly arises in a nodular goitre or a foetal adenoma. Histologically, carcinomata of the thyroid may be subdivided into several types ranging from extremely anaplastic tumours (carcinoma simplex) to well-differentiated adenocarcinomata. Some tumours metastasize mainly to the jugular lymph nodes; others metastasize at an early stage by the blood stream, especially to the lungs and bones.

Clinically, the tumour may take the form of a diffuse, hard, fixed swelling or a localized nodule. With diffuse growths dyspnoea occurs early, partly due to direct pressure on the trachea and partly to involvement of the recurrent laryngeal nerves. With the localized type of growth the diagnosis of carcinoma may be suggested by the hardness of the swelling or by its rapid increase in size; sometimes, however, the primary tumour is so small as to be impalpable and its existence is unsuspected until metastasis occurs.

*Treatment.* Every localized swelling in the thyroid should be removed without delay. In many cases the lesion will be found on histological examination to be benign; if it proves to be malignant the patient may be given post-operative treatment in the form of X-ray therapy, administration of thyroxine or administration of radioactive iodine, depending on the

type of tumour. Provided it is still encapsulated and has not metastasized there is quite a good chance of cure.

If the patient is not seen until the diagnosis of carcinoma of the thyroid is obvious, treatment is difficult and the outlook is poor because the growth has usually become fixed to other structures in the neck or has metastasized by the blood stream. In such cases supervoltage X-ray therapy, or surgical excision of as much of the gland as possible followed by administration of radio-active iodine, offers the best hope.

### **Thyroid deficiency (Hypothyroidism)**

Congenital absence or mal-development of the thyroid gland results in *cretinism*. Thyroid deficiency developing later in life causes *myxoedema*. These conditions are described in textbooks of medicine.

## **The Parathyroids**

### **Hyperparathyroidism**

Excessive secretion of parathyroid hormone causes *hyperparathyroidism*. This may be *primary* or *secondary*. Primary hyperparathyroidism is due usually to an adenoma arising in one gland, but occasionally to generalized hyperplasia or to a parathyroid carcinoma. Secondary hyperparathyroidism occurs as a consequence of advanced renal disease.

Primary hyperparathyroidism may lead to pathological changes in the urinary tract (in the form of calculus formation, deposition of calcium in the renal parenchyma, or sometimes renal failure without evidence of pathological calcification), or in the skeleton (in the form of widespread decalcification or generalized osteitis fibrosa cystica—Chapter XIX), or in both. It may also cause dyspepsia.

The level of serum calcium is raised, and that of the serum inorganic phosphorus is lowered. An adenoma, if present, is almost never palpable on examination of the neck.

The only effective treatment is surgical excision of the affected parathyroid. Even at operation the tumour may be difficult to find.

### **Parathyroid deficiency**

Parathyroid deficiency sometimes follows the operation of thyroidectomy, as a result of inadvertent removal or devascularization of parathyroid tissue. The serum calcium is lowered and this leads to a condition known as *tetany*, characterized by hyperexcitability of skeletal muscles.

Symptoms usually begin within 48 hours of operation; they include headache, restlessness, increased pulse rate and twitching of muscles.

Characteristic spasms occur in the hands in which the fingers are flexed at the metacarpo-phalangeal joints and the thumbs are adducted; similar spasms may occur in the feet. The facial muscles can often be made to twitch by tapping over a branch of the facial nerve (Chvostek's sign). In severe cases there may be difficulty in breathing owing to spasm of the muscles of the larynx.

The essential treatment is to bring the lowered serum calcium back to normal. This can be achieved by giving intravenous injections of calcium gluconate. In many cases treatment can be stopped after a few days. If the condition persists the patient should be given calciferol (vitamin D) or dihydrotachysterol as well as calcium. Parathormone is not used for long-term treatment because it has been reported that it ceases to be effective after a few weeks, possibly because the patient forms an antibody against it.

### **Other Endocrine Glands**

It will not be necessary to consider other endocrine glands in detail, but a few facts may be mentioned.

*Tumours or hyperplasia of the suprarenal cortex* cause a variety of syndromes including *Cushing's syndrome* (*vide supra*), the *adrenogenital syndrome* and *aldosteronism*, depending on the type of hormone which is secreted in excess. Surgical treatment is indicated, but it is often impossible to decide clinically whether the left adrenal, or the right or both are involved. Frequently, therefore, both have to be exposed at operation though they are not necessarily both removed. If both are removed completely the patient must be maintained permanently on cortisone.

*Suprarenal cortical deficiency* causes a condition known as Addison's disease, characterized by weakness, low blood pressure, gastrointestinal disturbances and skin pigmentation. The underlying lesion in about fifty per cent of cases appears to be destruction of the adrenals by tuberculosis; in many of the remainder the condition is probably a form of autoimmune disease. The prognosis used to be poor, but nowadays nearly all patients can be maintained in good health by administration of cortisone combined if necessary with increased intake of sodium chloride. In cases thought to be due to tuberculosis appropriate antibiotic therapy may also be given.

Deficient secretion by the islets of Langerhans in the pancreas results in *diabetes mellitus*, a serious disorder of metabolism which is fully described in textbooks of medicine. The diagnosis is suggested by finding sugar in the urine and confirmed by studying the changes in the blood sugar following administration of 50 grammes of glucose by mouth.

Diabetics have a lowered resistance to infection and are liable to develop diabetic gangrene. They are poor anaesthetic risks and need special pre-operative and post-operative treatment; for this reason the urine should always be tested for sugar before an operation is performed under general anaesthesia.

*Excessive secretion of insulin* is a rare condition, but occurs occasionally as the result of an adenoma arising in pancreatic islet tissue. The blood sugar is lowered and the patient is liable to recurrent attacks of coma. The immediate treatment in an attack is to inject glucose solution intravenously; for permanent cure surgical excision of the adenoma is required.

## *Chapter XXIII*

# LESIONS CAUSING OBSTRUCTION OF DUCTS AND HOLLOW VISCERA

Many lesions owe their surgical importance wholly or partly to the fact that they cause obstruction of the alimentary, respiratory or urinary tract, or of the duct of a gland. Despite the wide diversity of these lesions it will be convenient to consider them as a group.

### **Causes of Obstruction**

Obstruction may be caused by a mechanical block. In the alimentary and urinary tracts it may also be caused by interference with the neuro-muscular mechanism responsible for the onward passage of material.

#### **Obstruction due to mechanical block**

The following types of lesion may be responsible:

1. *Abnormal material within the lumen of the tube.* Common examples are urinary, biliary and salivary calculi, and foreign bodies such as dentures and tooth fragments which have been swallowed or inhaled.
2. *A lesion in the wall of the tube causing narrowing of the lumen.* Examples are congenital stenosis, and stricture due to chronic inflammation or malignant disease.
3. *Pressure on the tube from without.* Common examples are constriction of the gut in a hernial sac, and pressure on the trachea due to a goitre.
4. *Twisting of a segment of the tube.* This condition is known as a volvulus. It occurs most commonly in the colon.

#### **Obstruction due to disturbance of the neuro-muscular mechanism**

Obstruction due to disturbance of the neuro-muscular mechanism may arise in the following ways:

1. *Muscular paralysis.* A good example is the condition known as *paralytic ileus* in which the smooth muscle of the gut becomes paralysed, peristalsis ceases and the gut becomes passively distended. Paralytic ileus is most commonly due to peritonitis.

Another example is paralysis of the gut due to interference with its blood supply, caused by thrombosis or embolism of the mesenteric vessels.

Paralysis may also supervene in cases of long-standing mechanical obstruction as a result of fatigue.

2. *Muscle spasm*. The spasm may be confined to a specialized sphincter or may occur over a considerable length of the tube. A common example of the former type is spasm of the pylorus due to an active duodenal ulcer; an example of the latter type is bronchial asthma, a condition in which respiratory difficulty occurs as a result of widespread spasm of the smooth muscle of the bronchi.

3. *Achalasia*. This term is applied to conditions in which a sphincter fails to relax at the proper time but in which there is no true spasm; that is, the *resting* tone of the sphincter is not increased. Achalasia of the cardia, which is described later, is the most important example.

### Effects of Obstruction

The effects of obstruction depend on several factors, of which the most important are the site, the cause and degree of the obstruction, and the speed with which it develops.

Severe colicky pain is characteristic of mechanical obstruction, except in the later stages when muscular fatigue has led to paralysis and passive distension.

The fact that material is not passing normally along the obstructed tube may be obvious; in complete intestinal obstruction, for instance, absolute constipation occurs, and in obstruction of the common bile duct the stools become pale from lack of bile.

Regurgitation or vomiting is characteristic of obstruction of the alimentary tract, and if the obstruction is in the upper jejunum it is accompanied by severe toxæmia.

Obstruction of the duct of a gland, or of the urinary tract, leads to distension from back pressure. A retention cyst may develop or, in urinary obstruction, a condition known as *hydronephrosis* in which there is dilatation of the renal pelvis and calyces. Infection is likely to develop if the obstruction is not promptly relieved.

Other possible sequelæ are the development of a fistula (Chapter XVI) and the formation of calculi. A *fistula* is most commonly the result of an unsuccessful attempt to treat the obstruction by operation. It relieves the back pressure, but may itself be a source of trouble. It is unlikely to close until the obstruction has been relieved. *Calculus formation* occurs mainly in the urinary and biliary tracts, and in the salivary glands and their ducts. A calculus may be either the cause or the result of obstruction.



Above the site of obstruction material may be reabsorbed into the blood stream. In obstruction of the common bile duct, for example, jaundice is produced by reabsorption of bile pigments.

Obstruction of the respiratory tract presents special problems; these are discussed below.

### **Principles of Treatment**

The fundamental principle of treatment is to remove the cause of the obstruction whenever possible.

In *mechanical obstruction* stones or foreign bodies in the lumen of the tube are removed, and strictures are treated by dilatation, by plastic operations, or by resection of the affected segment of tube followed by anastomosis to restore continuity. If curative procedures of this sort are not possible a short-circuiting operation may be performed. Sometimes an external fistula is deliberately created above the level of the obstruction; a common example is the creation of a *colostomy* (artificial anus) above an obstruction in the large bowel. Subsequently it may be possible to remove the obstruction and close the fistula, but with a lesion which cannot be removed, such as an inoperable carcinoma, the fistula will be permanent.

In *achalasia* simple dilatation of the sphincter may suffice. If this fails, operative treatment may be tried. The operations performed are of three kinds:

1. Simple division of the sphincter muscle.
2. Plastic operations on the sphincter.
3. Division of nerves which normally cause constriction of the sphincter.

So far we have dealt only with general principles. It remains to illustrate these principles by specific examples.

### **Respiratory Obstruction**

It will be convenient to consider obstruction at four levels in the respiratory tract.

#### **The nose and naso-pharynx**

In children it is not uncommon for a foreign body to become impacted in the nose. There is no serious interference with respiration and the condition may remain unsuspected until a chronic unilateral nasal discharge develops. Removal of the foreign body is usually easy, but in young children a general anaesthetic should be given.

Chronic obstruction of the nasal passages is an important contributory cause of infection of the accessory sinuses (Chapter X). The usual causes of obstruction are deviation of the septum, which may be congenital or traumatic; hypertrophy of the turbinate bones; and nasal polypi. Appropriate surgical treatment is required in each case.

Chronic obstruction of the naso-pharynx in children commonly occurs as a result of hypertrophy of masses of lymphoid tissue known as the *adenoids*. The condition leads to mouth breathing and this in turn may cause mal-development of the teeth. Surgical removal of the adenoids by curettage is required.

### **The larynx**

A foreign body may become impacted at the entrance to the larynx or just above the vocal cords. Such an accident is especially liable to occur under anaesthesia, and proper precautions must be taken to avoid it. Dentures should always be removed before an anaesthetic is given, and with operations in the mouth the pharynx should be packed off to prevent the inhalation of blood or foreign bodies such as tooth fragments.

Laryngeal obstruction causes urgent dyspnoea, and if the obstruction is not relieved death may occur within a few minutes. The foreign body may sometimes be hooked out with a finger; if this fails it should be removed under direct vision through a laryngoscope. If a laryngoscope is not available an attempt should be made to dislodge the foreign body by turning the patient upside down.

During anaesthesia respiratory obstruction may also occur from the tongue falling back and blocking the entrance to the larynx, or from spasm of the glottis. The former condition is treated by the simple manoeuvre of pulling the tongue and the jaw forward. Spasm of the glottis is more troublesome; it usually occurs during the induction stage and is especially likely in patients with septic lesions in the neck such as Ludwig's angina (Chapter X). In these cases it is particularly dangerous to use intravenous anaesthetics like thiopentone. The spasm usually relaxes sufficiently to allow a tube to be passed through a laryngoscope, but in one case of this kind the writer had to perform an emergency tracheostomy (*vide infra*).

Other causes of acute laryngeal obstruction are laryngeal diphtheria (Chapter XII), 'oedema of the glottis' (Chapter X) and division of both recurrent laryngeal nerves. The last condition may occur as an accident during the operation of thyroidectomy (Chapter XXII). The patient's life depends on prompt relief of the obstruction and this usually necessitates tracheostomy. The operation is described below.

Obstruction which develops more slowly may result from laryngeal

palsy caused by pressure on the recurrent nerves, or occasionally from tuberculosis, syphilis or neoplastic disease of the larynx. Tracheostomy may be required if progress of the condition cannot be arrested.

#### *Emergency tracheostomy*

Occasionally a patient's life may depend on the ability of a dental surgeon to perform emergency tracheostomy; every dental surgeon should therefore be familiar with the steps of the operation.

The patient is placed on his back with his head extended over a small pillow or rolled-up towel. The important landmarks in the mid-line of the neck are palpated; from above downward they are the point of the chin, the hyoid bone, the thyroid cartilage (Adam's apple), the cricoid cartilage and the *manubrium sterni*. If the patient is conscious and time permits, the tissues in the line of the proposed incision are infiltrated with novocaine; in an unconscious patient, or in case of desperate urgency, this step is omitted. The trachea is steadied with the left thumb and index finger and an incision is made exactly in the mid-line, extending downwards for 2 inches from the cricoid cartilage (Fig. 102). The skin and subcutaneous fat are divided and the incision is deepened in the interval between the two sterno-hyoid muscles until the cartilaginous rings of the trachea can be felt or seen. The second, third and fourth rings are divided in the line of the incision, the opening in the trachea is dilated with forceps and a tube



FIG. 102. Incision for emergency tracheostomy.

is inserted. The isthmus of the thyroid crosses the trachea in this region; it may be displaced downwards or divided between forceps. A proper metal tracheostomy tube having a removable inner tube should be used if available; if necessary the wound in the trachea may be temporarily held open with forceps until a tube is obtained. Care must be taken to see that the tube is inserted into the lumen of the trachea and not between the trachea and the fascia overlying it. During the operation bleeding vessels are clamped with artery forceps and ligated; provided the surgeon keeps to the mid-line haemorrhage is rarely troublesome.

When the patient requires artificial respiration with a mechanical respirator a rubber tube with an inflatable cuff is used instead of a metal tube.

### **The trachea**

Obstruction of the trachea is usually due to pressure from without and the commonest cause is enlargement of the thyroid gland (Chapter XXII). The obstruction usually develops gradually, but may occur suddenly as a result of haemorrhage into a cyst. With acute obstruction emergency tracheostomy may be necessary; in less urgent cases the treatment is directed to the underlying cause.

### **The bronchi**

Obstruction of the smaller bronchi, due to muscle spasm, occurs in bronchial asthma. The patient experiences great distress and difficulty in breathing, especially with expiration. The immediate treatment in an attack of asthma is to give a subcutaneous injection of adrenalin. The condition is fully described in textbooks of medicine.

Sudden obstruction of a main bronchus may occur from mechanical block due to impaction of a plug of mucus or an inhaled foreign body such as a tooth or tooth fragment. Obstruction by a plug of mucus occurs as a complication of operations and is the cause of post-operative pulmonary collapse. The symptoms and treatment of this condition have been described in Chapter II. An inhaled foreign body impacted in a bronchus should be removed through a bronchoscope; if it is not removed a lung abscess may develop.

Primary carcinoma of the lung usually originates in a bronchus and is much the commonest cause of gradual bronchial obstruction. The initial symptoms may be slight, but later dyspnoea, severe pain in the chest, and coughing of blood-stained sputum develop. Pulmonary collapse develops in the area of lung distal to the block and is responsible for the physical signs found on examination of the chest.

Diagnosis is confirmed by bronchoscopic examination and biopsy.

The only treatment of value is removal of the affected lobe (*lobectomy*) or lung (*pneumonectomy*). In early cases the results are encouraging.

### **Dysphagia**

Dysphagia means difficulty in swallowing. It results from mechanical block or a neuro-muscular disorder in the pharynx or oesophagus.

#### **Dysphagia due to disorders of the pharynx**

The usual causes are as follows:

1. *Mechanical block*

Pharyngeal diverticulum.

Foreign bodies.

Malignant disease.

2. *Neuro-muscular disorders*

Pharyngeal paralysis.

Plummer-Vinson syndrome.

Globus hystericus.

#### *Pharyngeal diverticulum*

The typical pharyngeal diverticulum (Fig. 103) occurs in middle-aged or elderly men. It arises in the mid-line posteriorly, at the junction of the pharynx and oesophagus. Dysphagia gradually develops because food enters the pouch and causes it to become distended and exert pressure on the oesophagus. Regurgitation is common, but some food remains in the



FIG. 103. Radiograph taken after a barium swallow showing a pharyngeal diverticulum.

pouch and undergoes decomposition. Very occasionally the pouch is palpable as a swelling in the neck.

The diagnosis is suggested by the history and confirmed by radiography after giving the patient a barium swallow (Chapter I).

Treatment is by operation, and the usual procedure is to remove the pouch.

### *Foreign Bodies*

Foreign bodies of sufficient size to cause complete obstruction rarely become impacted in the pharynx, but occasionally the condition arises from swallowing a denture. The main symptom is urgent dyspnoea. Small objects such as fish bones often get caught in the pharyngeal mucosa and cause dysphagia owing to the pain they produce. These small foreign bodies should always be removed through an oesophagoscope, otherwise they lead to infection which may be complicated by the development of a fatal mediastinitis.

### *Malignant disease*

Malignant disease of the pharynx has been described in Chapter XIV. The tumour which usually causes dysphagia is post-cricoid carcinoma, a growth which occurs almost exclusively in women.

### *Neuro-muscular disorders*

*Pharyngeal paralysis* occasionally results from diphtheritic paralysis and other diseases of the nervous system.

The *Plummer-Vinson syndrome* occurs in middle-aged women, and is characterized by dysphagia, anaemia and usually achlorhydria. The dysphagia is due to achalasia of the muscles at the junction of the pharynx and oesophagus. The anaemia usually responds to treatment with iron and the dysphagia often improves as the level of haemoglobin rises.

*Globus hystericus* is a functional disorder (Chapter XXI) in which the patient complains of dysphagia accompanied by a feeling of a lump on the throat. This condition must never be diagnosed until organic causes of dysphagia have been excluded.

## **Dysphagia due to disorders of the oesophagus**

The usual causes are as follows:

### *1. Mechanical block*

Carcinoma of the oesophagus.

Cicatricial stenosis.

Diaphragmatic hernia.

## 2. *Neuro-muscular disorders*

### *Achalasia of the cardia.*

#### *Carcinoma of the oesophagus*

Carcinoma of the oesophagus has been described in Chapter XIV. The differential diagnosis from achalasia of the cardia is discussed below.

#### *Cicatricial stenosis*

The term cicatricial stenosis is applied to a stricture of the oesophagus resulting from injury or inflammation. The commonest cause is chemical injury; that is, swallowing corrosive fluids. The stricture may develop slowly over a period of months or years.

The main symptoms are dysphagia and wasting.

The degree and extent of the stricture are determined by a barium swallow. In some cases treatment by gradual dilatation is effective; failing this the choice lies between operative resection of the stricture and gastrostomy (that is, the creation of a permanent opening into the stomach for feeding the patient).

#### *Diaphragmatic hernia*

Diaphragmatic hernia is a condition in which, owing to a congenital defect or an injury of the diaphragm, abdominal viscera are displaced upwards into the chest. When part of the stomach is situated above the diaphragm the patient may experience attacks of retrosternal pain and regurgitation of food, followed later by dysphagia. These symptoms are caused by the backflow of gastric juice into the oesophagus with consequent ulceration and stenosis. The diagnosis is made by barium swallow. The treatment of choice is operative reduction of the hernia, with resection of the stenosed portion of oesophagus if required. Sometimes the oesophagus is abnormally short and operative treatment may then be impracticable.

#### *Achalasia of the cardia*

In achalasia of the cardia the sphincter muscle at the junction of the oesophagus and the cardiac part of the stomach fails to relax when a peristaltic wave reaches it; food is therefore prevented from entering the stomach and the oesophagus becomes distended and hypertrophied. There is no true spasm of the sphincter, but the term *cardiospasm* is often erroneously used to describe the condition.

Achalasia of the cardia may occur in either sex. The main symptoms are dysphagia, regurgitation of food and loss of weight; they usually begin in early adult life or middle age.

The differential diagnosis from carcinoma of the oesophagus is based on the following observations:

1. *The age of the patient.* Symptoms of achalasia usually begin between 20 and 40; carcinoma is uncommon before 50.

2. *The character of the dysphagia.* In achalasia the dysphagia may be intermittent; when it is present fluids are usually as difficult to swallow as solids. In carcinoma the dysphagia is steadily progressive and difficulty in swallowing fluids occurs only in the late stages.

3. *The character of the regurgitated material.* In achalasia the fluid regurgitated is turbid but not usually foul-smelling; in carcinoma it is foul-smelling and may contain blood.

4. *The radiological appearances after giving a barium swallow.* In achalasia the upper part of the oesophagus is enormously dilated and the lower part is smooth and conical. The obstruction is situated at the junction of oesophagus and stomach (Fig. 104). Carcinoma may occur at any level. There is irregular narrowing of the oesophagus with only slight dilation above (Fig. 105).



FIG. 104



FIG. 105

FIG. 104. Radiograph taken after a barium swallow in a patient with cardiospasm.

FIG. 105. Radiograph taken after a barium swallow in a patient with a carcinoma of the oesophagus.



5. *Oesophagoscopy*. In achalasia the oesophagus contains much turbid fluid; when this has been sucked out the instrument can be passed into the cardia without difficulty. In carcinoma the growth can be seen and a piece can be removed for histological examination.

*Treatment*. The patient is taught to swallow mercury-loaded rubber tubes of gradually increasing size. This simple treatment is often effective, but sometimes operation, with division of the circular muscle at the junction of oesophagus and stomach, is needed.

### **Salivary Calculus**

Salivary calculi are composed of calcium carbonate and calcium phosphate mixed with organic material. They occur commonly in the submaxillary gland or duct but are rare in the parotid; this is probably because the parotid gland has a purely serous secretion whereas the submaxillary gland also secretes mucus.

A stone in the submaxillary duct causes intermittent swelling of the gland. At times of inactivity only a small amount of saliva is formed and this can leak past the stone; when secretion is stimulated by food, however, saliva is dammed back and causes pain and swelling of the gland. If the obstruction is not relieved infection occurs behind it; this may lead to suppuration and secondary calculus formation in the gland itself.

The diagnosis is suggested by the history, and is confirmed by seeing the stone at the orifice of the duct or feeling it on bimanual palpation, or by X-ray (Fig. 106).



FIG. 106. Radiograph showing a calculus in the posterior part of Wharton's duct.

Stones in the gland proper give less definite symptoms. There is usually dull pain and gradually increasing swelling of the gland, and pus may enter the mouth through the corresponding duct.

*Treatment.* A stone in Wharton's duct can usually be removed from within the mouth by incising the wall of the duct, or by enlarging its orifice if the stone is visible. This operation is usually performed under local anaesthesia. If the submaxillary gland is infected or contains calculi it should be removed through an incision in the neck.

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